

Obstetrics and Gynaecology

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Subject: **Obstetrics and Gynaecology**



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25/4/18

OBS & GYNAE

(1)

RELEVANT ANATOMY

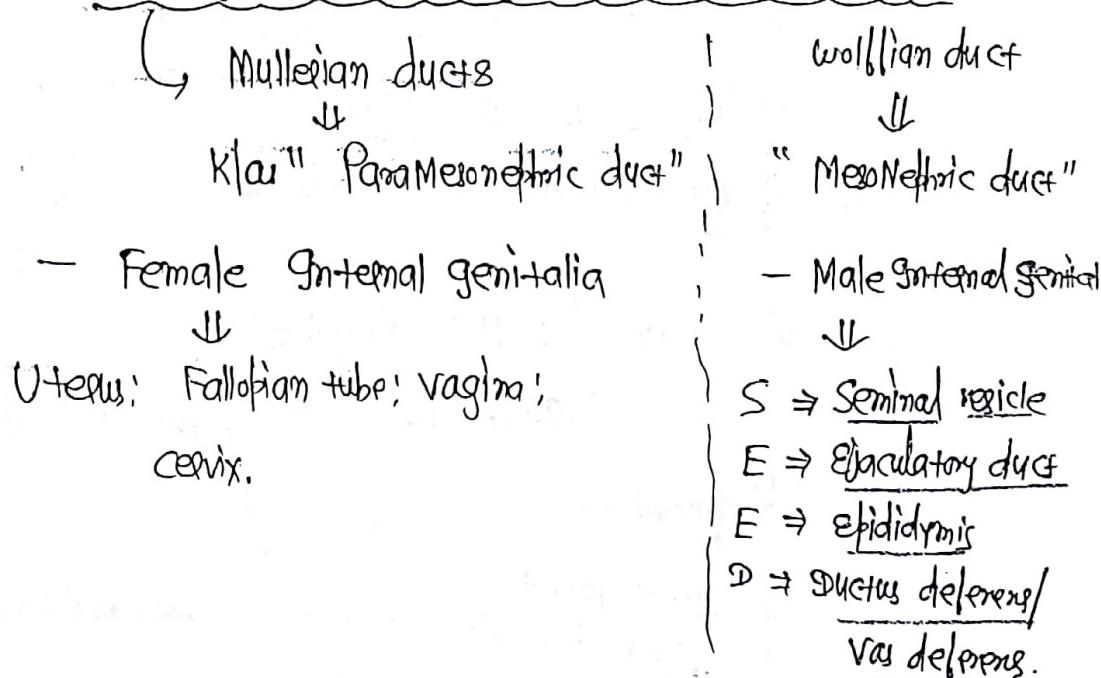
GONADS

Internal genitalia

External genitalia

Internal genitalia \rightarrow Uterus; cervix; Fallopian tube; vagina

embryological structure from which it develops \rightarrow



* Both the ducts appear in every foetus \oplus 6 weeks.

* Mullerian duct is lateral to Wolffian duct, \downarrow either ♂ or ♀

* Mullerian disappears \oplus 9 weeks / Wolffian disappears in ♀.

Mullerian ducts

Disappear \Rightarrow In Male

Why?



Testis



MIS (Mullerian Inhibiting Substance)

* gives testes

Released by "Sertoli cells"

↳ Start producing
@ 2 weeks

- Mullerian duct will present In female b/c of Absence of MIS

* Estrogen is Not Required for development of uterus, vagina, etc; for it absence of testosterone

Required.

Papophoron \rightarrow medial part of broad ligament

Epoophoron

Gartner's duct

Hydatid of Morgagni

Klaf's "organ of Rosenmüller"

Lateral Part of Broad Ligament

all contents of broad ligament

* Remnants of Wolffian duct

In Female

i) Epoophoron - Cranial Remnants of Meometriac tubules

ii) Papophoron - Caudal Remnants of Meometriac tubules

iii) Gartner's duct Caudal Remnants of Meometriac ducts

* Gartner's duct Sometimes forms a cyst in the vagina

* Gartner's cyst ⇒ In Anterior Lateral wall of vagina (2)

♀/♂ → Bartholin's cyst ⇒ In Posterior Lateral wall of vagina

M/c cyst of vagina/vulva ⇒ Inclusion cyst

Located @ Lateral wall.

* Remnant of Mullerian duct in Males ⇒

While Appendix of testis = K/a. "Hydatid of Morgagni".

Appendix of epididymis is Remnant of Wolffian duct.

* Mullerian duct ⇒ ♀ In No.

both ducts fuse & form uterus in 10 weeks

We can differentiate External genitalia @ 10 weeks

→ direction of Fusion — Fusion begins from centre then it moves cranially/caudally.

Direction is from caudal to cranial. (U)

- * Initially Ovary is solid organ; Later cavity formation
occur @ 18-20 weeks
 - by dissolution of a midline fibrous septum
 - Potential cavity
 - ↳ to accommodate something

- * If Fundus is convex upwards ⇒ No fusion Abnormality
- If Fundus is dipping ⇒ Fusion Abnormality seen.

- * complete Septate defect ⇒



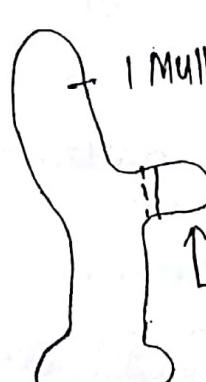
- * Incomplete septate defect ⇒

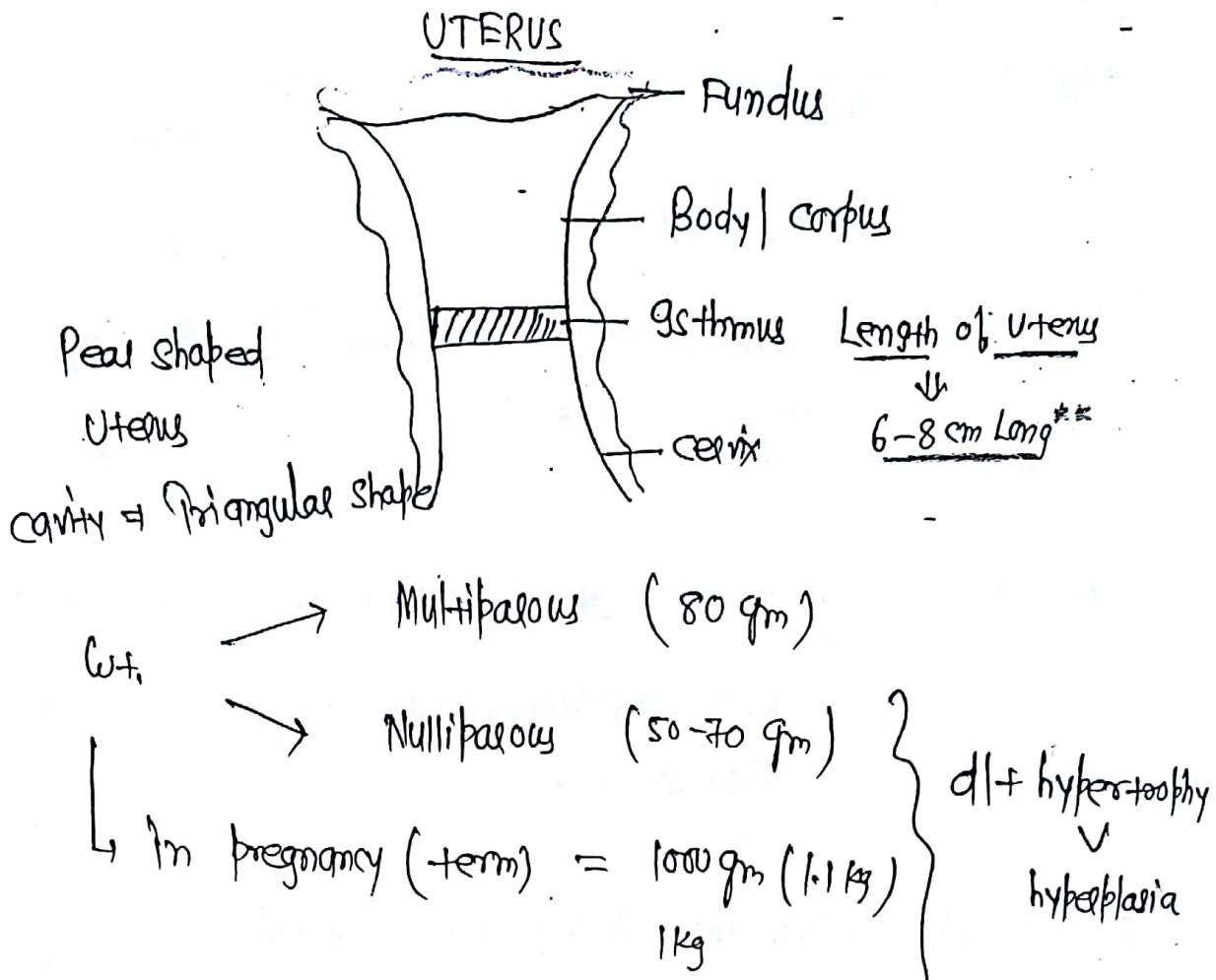


- * M/c Mullerian Anomaly ⇒ Septate Anomaly
 - * 2nd M/c Mullerian Anomaly ⇒ Bicornuate Uterus (Bicornuate unicollis) (Bicornuate bicollis)
 - * M/c Mullerian Anomaly alw abortion
 - * M/c Mullerian Anomaly alw Infertility
 - * Worst Reproductive outcome
- two uterus one cervix
↑
Bicornuate unicollis
V
Bicornuate bicollis
↑
two uterus two cervix

- * In Diadelphic uterus both Mullerian duct form Anomaly
(Complete Lack of Fusion)
 - ↳ complete failure of Fusion
 - Vaginal Septum

- * Mullerian Anomaly & vaginal septum \Rightarrow Didelphys (3)
- * Bicornuate Uterus has good Reproductive outcome
 - \hookrightarrow What pregnancy complication? \downarrow Pre-term Labour if Most Likely to do Abortion
- * Didelphys Uterus also has good Reproductive outcome
 - \hookrightarrow What pregnancy complication? \downarrow Pre-term Labour if Most Likely to do
- * Usually Corrective Surgery is Not Required
 - \hookrightarrow If we want then do Unification surgery

"STARRSMAN METROPLASRY"
- * Unicornuate Uterus \downarrow
 - 1 Mullerian duct = develops \uparrow May cause ectopic
Communicating
 - 2nd Rudimentary \downarrow Non-communicating \downarrow May cause
Severe dysmenorrhea
(Unilateral dys-
Menorrhea)

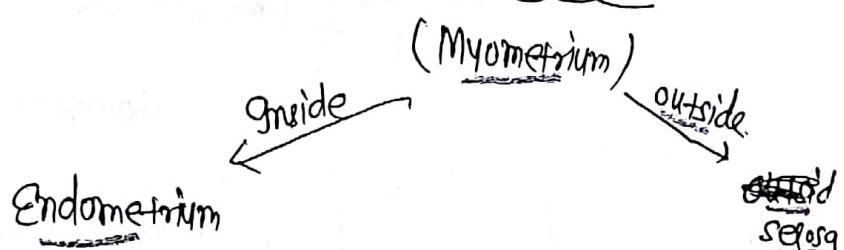


Wt. → Multiparous (80 gm)
 ↓ → Nulliparous (50-70 gm) }
 In pregnancy (term) = 1000 gm (1.1 kg) }
 1kg }
 dl + hypertrophy
 v
 hyperplasia

volume of Non-pregnant = 10mL } dl + hypertrophy >> hyperplasia
 volume of Pregnant = 5L }

Q.Q Weight of Uterus six week after Postpartum = 80 gm

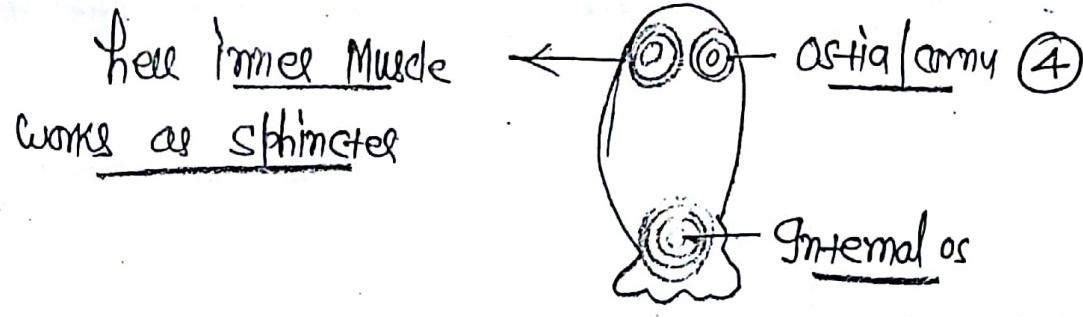
* Body ⇒ Is Made up of Smooth Muscle



- Myometrium = 2-2.5 cm thick

3 Layer ⇒

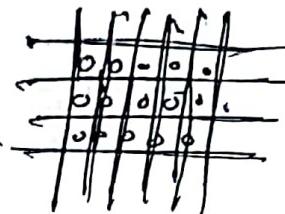
- 1. Inner - circular Muscle
- 2. Middle - criss-cross
- 3. Outer - Longitudinal



⇒ Middle Layer = Criss-cross



work as Living Ligature *



⇒ Endometrium

↳ Gland + Stroma



Simple tubular

↳ Single Layer Columnar epithelium

cilia prst. only Near gland opening

↳ Superficial Layer = Functional ⇒ Shed off every Month
in Menstruation

Basal Layer = Not shed off

↳ Function = Regeneration

↳ So thickness of endometrium changes

Just after Menstruation (D_5) =

Thickness
0.5 mm

Periovulatory = 2-3 mm

Semenary = 5-6 mm

Implantation = 10-12 mm

vigorous curettage \Rightarrow damage the basal layer



Result in Intrauterine Adhesion



Result in

Asherman's Syndrome

~~Asherman's Syndrome~~

Post partum hemorrhage

the ~~Post partum hemorrhage~~

~~Pt. is with Amenorrhoea infertility~~

~~Infertility~~

Pt. is with Amenorrhoea infertility

Highest Risk in to Manage

\Rightarrow Outside part of Myometrium = Serosa

Anteriorly Loose fold of Peritoneum \Rightarrow Uterovesical fold

Posteriorly Loose fold of Peritoneum \Rightarrow Rectouterine Pouch

* At what Level Cervix opens -
- Into cervix \rightarrow Internal os

Anatomical (Above)

0.5 cm
 \downarrow

Histological (Below) \downarrow constriction

Kl" gsthmus

* Gsthmus is b/w Anatomical & Histological Internal os.

* Gsthmus — Stretch — LUS

\hookrightarrow Lower Ateline segment

In term LUS Gsthmus + cervix ; cervix comes above utr

(70%) (30%)

Cervical attachment (taking up of cervix).

Cervical effacement

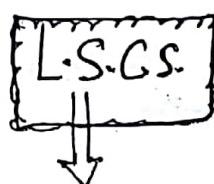
(5)

↳ Shortening + softening

* At term LUS \Rightarrow 5cm

In complete (Manual Labor) LUS = 10cm

* LUS shortly forming after 1st Trimester.



At Lower Segment

How to
Identify?

by Loose fold
Peritoneal



Most common Incision \Rightarrow Low Transverse Incision



Kerr's Incision

Shape \Rightarrow Transverse or Pfannenstiel
Klas "Kerr's Incision"



Kronig's Incision

Low Vertical Incision

Klas "Kronig's Incision"



Classical Incision

Classical Incision

Klas "Classical Incision"

Risk of Rupture

Risk of Rupture

- * Low transverse Incision \Rightarrow 0.2-0.9%
- * Low vertical Incision \Rightarrow 1-7%
- * Classical Incision \Rightarrow 4-9%
↳ weakest Incision

* ~~⑤~~ If classical Incision scar is already given

↳ It is Absolute Indication for Repeat C-section
Section

but Not an indication for Repeat classical Incision

- * Indication of classical \Rightarrow ① \oplus in case;
② Dense Adhesion b/w bladder & Uterus (May injured bladder)
③ Repaired vagino-vestical fistula
④ Post Mortem C.S.

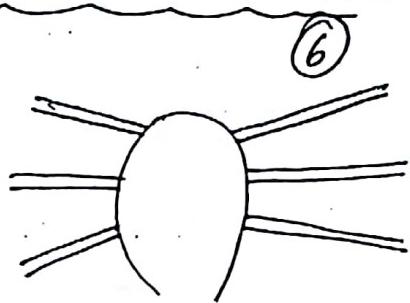
N.B.: Anterior Located Placenta Previa (only if a Not trained enough doctor)
↳ So; for Anterior Placenta Previa \Rightarrow LSCS do

* On other side of uterus 3 structures are attached:

Mnemonics

\downarrow
RTO

- Round Ligament
- Fallopian Tube
- Ovarian Ligament



Adnexa \Rightarrow Fallopian tube + ovary

Antero-Posteriorly \Rightarrow Round Ligament \rightarrow Fallopian tube

\downarrow
Ovarian Ligament

\downarrow
Utero ovarian Pedicle

Superio-Inferior \Rightarrow

Fallopian tube

\downarrow below it



Round Ligament / ovarian Ligament @ same Level

They assist you in tubal ligation surgery

\hookrightarrow M/c cause of failure of tubal

Ligation \Rightarrow Ligation of wrong structure

FUNDUS → Part of uterus lies above the attachment of Fallopian tube

Round Ligament Path ⇒ Upper uterus → deep inguinal ring

Pulling uterus anteriorly to in Anteverted position.

Inguinal canal

* CANAL OF NUCK (Absent in fetus only)

Fold of Peritoneum in fetus that contains Round Ligament & extends into inguinal canal.

Superficial Ring

It carries Round Ligament.

Inverted on Labia Majora

* Round Ligament

Ovarian Ligament

} developed from Gracilis muscle



Proximal ⇒ Ovarian Ligament

Distal ⇒ Round Ligament

Not derived from Müllerian duct.

Blood Supply

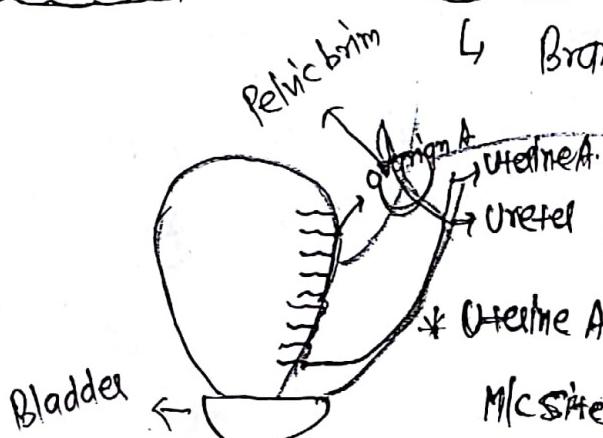
⇒ Uterine A

↳ Branch of Anterior division of Internal Iliac Artery.

2nd M/C site of ureteric injury

"Catched under bridge" ↳ Dangled Area
It is crossing over the ureter

M/C site for ureteric injuries



* Location of "water under bridge" Area \Rightarrow 2 cm lateral to cecum
OR
1.5 cm lateral to fornix.

* Ureters are posterior to ovary & uterine artery;
but it is anterior to internal iliac A 7

\Rightarrow Branches of Uterine A \uparrow

U \Rightarrow Uterine A.



A \Rightarrow Arcuate branches \Rightarrow Supply outer 1/3rd of Myometrium

R \Rightarrow Radial branches \Rightarrow Supply inner 2/3rd of Myometrium

B $\xleftarrow{\text{Basal}}$ Supply Basal endometrium

S $\xleftarrow{\text{Spiral}}$ Supplies the Superficial / Functional endometrium

- Always do B/L Ligation & do @ the Level of Internal os.

* Uterine A gives a special branch \Rightarrow Sampsons A \Rightarrow for Round Ligament

- Nerve supply \Rightarrow T₁₀, T₁₁, T₁₂, L₁

\hookrightarrow Pain during uterine contraction

travels via this Root

\hookrightarrow Pain Relay via "Frankenhauer ganglion."

We give "Labour Analgesia" (Level of block for vaginal delivery)

\hookrightarrow via Epidural Anaesthesia

\uparrow
T₁₀)

give "Bupivacaine"

\hookrightarrow "0.125 - 0.25%" (Dose)

\hookrightarrow Sensory block; if given rare
the motor block.

* Level of block for Cesarean Section

At T₄ (blk to knockout the peritoneal
Nerve Supply)

M/c Anesthesia \Rightarrow Spinal Anesthesia

* Labour Analgesia may Prolong Labour (Active phase by 1 hr)

↳ but doesn't ↑ Incidence of cesarean section,

* When we apply barrels (outlet/low) - Pudendal N. Block

done only when complete

dilatation head \oplus +3.

to block it if we

pierces Sacrospinous Ligament

S₂, S₃, S₄

↓

Previously K/o

Direction of Needle (Posterior Medial)

"Saddle block"

* Lymphatic drainage \Rightarrow Internal iliac + Ext. iliac L.N.

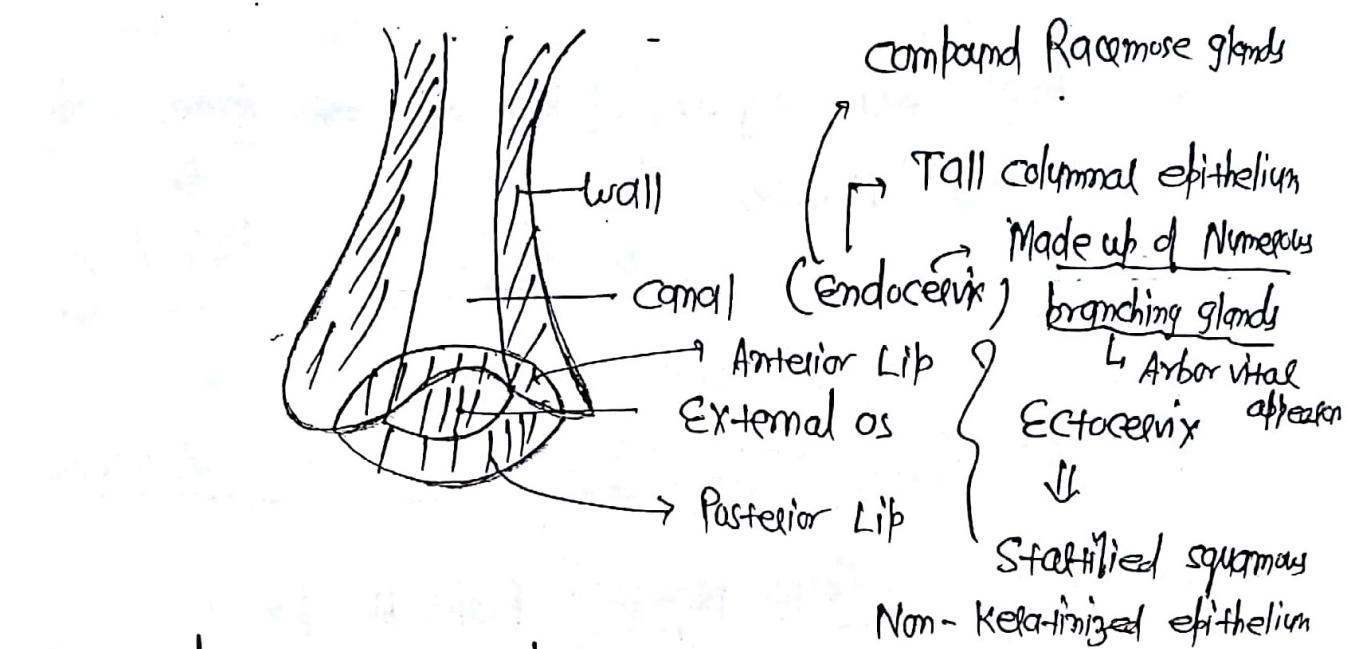
Fundus \longrightarrow Para-aortic L.N.

Ostia \longrightarrow Superficial inguinal L.N.

CERVIX \Rightarrow It opens into vagina * 15

- conical in shape
- 3cm Long
- cavity \Rightarrow spindle shape / fusiform

↓
⑧ @ external os **



External os \Rightarrow Shape

Nulliparous



Circular

Multiparous



Transverse slit

- Wall of cervix \Rightarrow Made up of connective tissue (collagen)

↓
10-15% Smooth Muscle

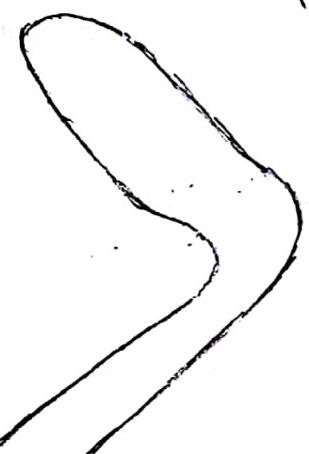
Result in effacement (there will be less
Softening & taking up) $\leftarrow \begin{cases} \text{In collagen \& to} \\ \text{In hyaluronic acid} \\ \& less water content \& less hydration \end{cases}$

* Broad Ligament is a potential space; contains blood vessels etc.

* Angle b/w cervix & vagina \Rightarrow Anteversion
 90° .

* Angle b/w Long axis of body of uterus & cervix \Rightarrow Anteflexion
 120° \hookrightarrow @ Internal os

* In 80% women Anteverted & Antiflexed uterus \oplus



Two Ligament Responsible for it

↳ Round Ligament
+
Uterosacral Ligament.

* If Fundus is More towards Bladder \Rightarrow Anteflexion

If Fundus is More towards Rectum \Rightarrow Retroflexion.

* In P/v examination \Rightarrow Which Lip you hitting 1st

↓
Anterior Lip
↓
Anteversion

↓
Posterior Lip
↓
Retroversion.

* Ratio of Cervix & corpus →

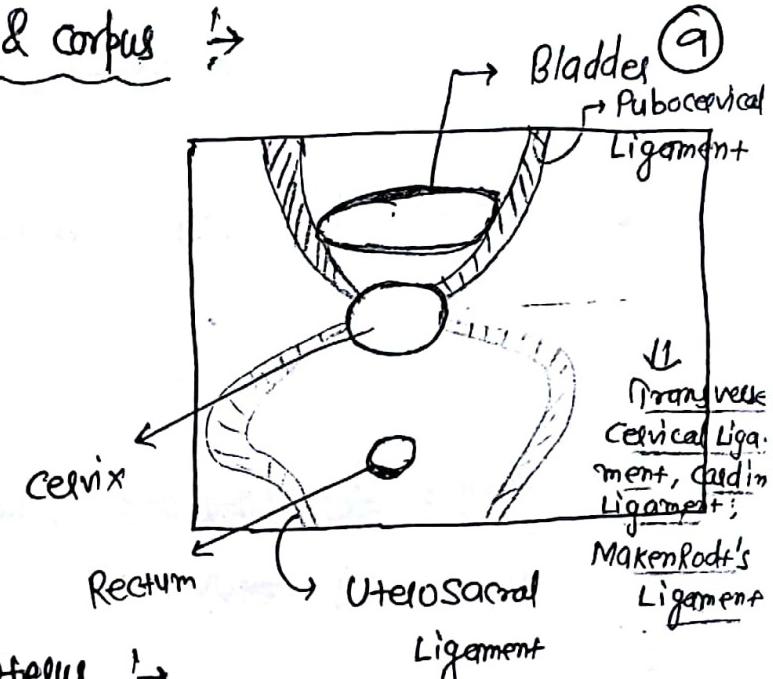
At birth = 1:1

Prepubertal = 2:1

after puberty = 1:2

Reproductive Life = 1:3

Post Menopausal = 1:1



* Main Support of Uterus →

Anterior ⇒ Pubocervical Ligament

Posterior ⇒ Uterosacral Ligament

Lateral ⇒ Piriformis | Cardinal | Mackenrodt's Ligament

Inferior ⇒ Levator Ani

Hammock Fibers
Radiate
Ligament = Pubocervical Ligament
Uterosacral Ligament
Cardinal Ligament.

Q All are Main Support except

↳ Round Ligament (Support, but Not Main)

← Broad Ligament (Not a support)

↳ False Name

It is Nothing, but fold
of Peritoneum

* Broad Ligament is a potential space; contains blood vessels etc.

* Angle b/w cervix & vagina \Rightarrow Anteversion
 \downarrow
 90° .

* Angle b/w Long axis of body of uterus & cervix \Rightarrow Anteflexion
 \downarrow
 120° \rightarrow @ Internal os

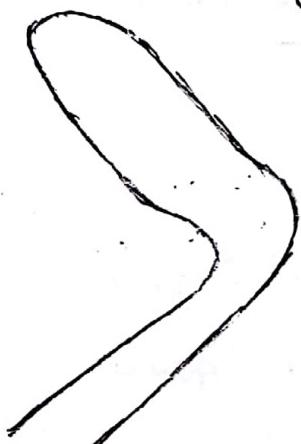
* In 80% women Anteverted & Antiflexed Uterus \oplus
 \downarrow

Two Ligament Responsible for it

↳ Round Ligament

+

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* In P/r examination \Rightarrow Which Lip you hitting 1st

↓
Anterior Lip
↓
Anteversion

↓
Posterior Lip
↓
Retroversion.

FALLOPIAN TUBE

- Unfused part of Mullerian duct*
- Length = 10 cm (10-12 cm)*

(10)

* Part of Fallopian tube (Medial - Lateral) :

- Intramural / Interstitial part (1-2 cm) = Narrowest part of Fallopian tube
- Uterine tube; (3 cm) = 0.7 mm diameter
- Ampulla; (5 cm) ⇒ Widest part of Fallopian tube = 6 mm
- Infundibulum
↳ Fimbrial end

Site of fertilization | M/c site of ectopic ♀

↓
bc here fertilization takes place & diff mucosal fold
at ② Ampulla (has "plicae")

* M/c site of ligation ⇒ Ectopic.

* Conceptus remains in Fallopian tubes ⇒ 3 days

* Conceptus enters the uterine cavity on the 4th day.
↳ Post-fertilisation

* Anatomical sphincter of Fallopian tube ⇒ Intramural part.

Physiological sphincter of FT ⇒ Ectopic

QQ Main Reason for transport of conceptus

↳ Tubal peristalsis *

Anything that lessens Tubal Motility Leads to "Ectopic ♀"

In Pelvic inflammatory disease;

Tubal Surgery ;

Progestrone only pills;

- epithelium of Fallopian tubes :

↳ Single Layer ciliated columnar epithelium.

3 Cells - Secretory

ciliated

Peg cells → Resting cells of Fallopian tubes.

* Direction of Ciliary Muscle is towards Uterus *

- Blood Supply ⇒ equal blood supply *

Medial 2/3rd ⇒ Uterine A.

Lateral 1/3rd ⇒ Ovarian A

↳ dilates 3 times in ♀

↳ site of Ligation for Management of PPV.

- Lymphatic drainage \Rightarrow Para-aortic Lymph Node*

(11)

IntraMural + ostia

\hookrightarrow Superficialinguinal Lymph Node*

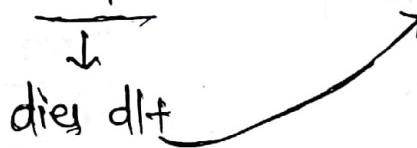
~~extra edge~~

N. Supply \Rightarrow

$T_{11} T_{12} L_1$

Pain sensation from Unruptured ectopic
(tubal stretch)

* Ectopic is Vascular accident



VAGINA **

Embryological development \rightarrow

Upper 1/3rd of Va

from Mullerian duct (Mesoderm)

Lower 2/3rd of vagina \Rightarrow

from Urogenital sinus (Endoderm)

\hookrightarrow from Sinovaginal

Hymen is Remnant of this \leftarrow bulb

* Mucous Membrane of vagina \Rightarrow from Endoderm of Urogenital sinus

* Muscles of vagina \Rightarrow from Mesoderm of Mullerian duct.

Vagina has four walls \Rightarrow Anterior



7-10 cm Long

Posterior

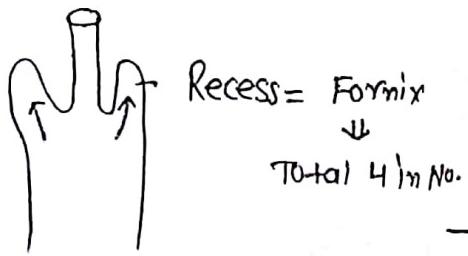
Lateral - 2 in No.

• Posterior wall is Longer than Anterior wall by 2 cm.*

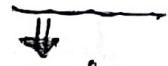
• A-P wall apposed together

↳ after cut "H" shaped

* Cervix comes inside vagina & Space b/w them is



"Formix"



4 in No. (Ant, Post, 2 Lateral)

- Posterior fornix is deepest*

if More than 100ml collection in Pouch of douglas \oplus

↳ it is significantly \uparrow

Pouch of douglas | Cul-de-Sac \Rightarrow Caudocentesis

Recto uterine fold Posterior
to Vagina

If we get blood - which doesn't clot

↳ Hemoperitoneum.

→ used in Ruptured ectopic.

If blood clot \Rightarrow then it comes from blood vessels.

Colpotomy \Rightarrow Opening of the Pouch of Douglas to drain Pelvic Pus. (Abscess) (12)

Enterocele \Rightarrow Prolapse of bowel wall (Pouch of Douglas later)

\hookrightarrow Upper 1/3rd of Posterior wall

Q. Which of the following is Cystocele?

\hookrightarrow Protrusion

a) Upper 2/3rd of wall;

Protrusion of bowel wall into the

b) Lower 2/3rd of wall;

Upper 2/3rd of Anterior wall of Vagina.

c) Upper 1/3rd of wall; * Rectocele \Rightarrow Prolapse into Middle 1/3rd of Posterior vaginal wall

d) Lower 1/3rd of wall \Rightarrow Urethrocele

\hookrightarrow Protrusion into Lower one-third of Anterior vaginal wall.

* \approx the H₃-line passing through Pelvis, vagina forms $\Rightarrow 45^\circ$

* Epithelium of vagina \Rightarrow Stratified Squamous Non-Keratinized

Secretion - Yes - Cervical secretion

Glands in vagina = No

comes as Transudative across vaginal wall.

&

Batholian glands only secrete during coitus

pH = Acidic \Rightarrow Natural defense Against infection

pH = Alkaline

Estrogen

Glycogen (Lactobacillus works) \rightarrow commensal

\approx presence of Glycogen

Doderleyn bacilli *

is brought about by Estrogen

Lactic acid (3.5-4.5) \Rightarrow (4-4.5) \Rightarrow It is natural defense of infection

Q.

Deficiency of 5 α Reductase \rightarrow

Male Pseudohermaphrodite (Genotype - Male)
Phenotype - Female

- * by looking external genitalia we differentiate Male & female by 12 weeks.

* Female Bartholin's gland Homologus Male Bulbourethral gland (Coper's gland)

* Glands of Skene Homologus Prostate.
(Para-urethral)

* Lymphatic drainage of clitoris \Rightarrow Superficial inguinal Lymph Node

* Lymphatic drainage of Glands clitoris \Rightarrow Deep inguinal Lymph Nodes
(Lymph Nodes of Cloquet)

* Lymphatic drainage of Labia Minora glands \Rightarrow Deep inguinal L.N.

* Lymphatic drainage of Labia Majora glands \Rightarrow Superficial inguinal L.N.

Vaginal foldBlood SupplyLymphatic drainage

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Upper 1/3rd

Descending Uterine Artery

External + Internal
Iliac

Middle 1/3rd

Inferior Vena Cava Artery

Internal Iliac

Lower 1/3rd

Middle Rectal Artery

Superficial Sanguinal

EXTERNAL GENITALIA

Genital tubercle → @ 6 weeks

FemaleMale (Homologous structure in Male)

Clitoris

Penis

II Fold

L. Minor

Penile urethra

II Swelling

L. Major

Scrotum

Absence of
DHT Required for
Female like organs.

To convert in Male form

Testosterone

↓ 5α-Reductase

↓ Dihydrotestosterone
↳ Pubic | Fold | Swelling.

Deficiency of 5α Reductase →

Male Pseudohermaphrodite (Genotype - Male)
Phenotype - Female

- * by looking external genitalia we differentiate Male & female by 12 weeks.

- | | |
|--|---|
| <p><u>Female</u></p> <p>* Bartholin's gland</p> <p>* Glands of Skene (Para-Urethral)</p> <p>* Lymphatic drainage of clitoris</p> <p>* Lymphatic drainage of Glands of clitoris</p> <p>* Lymphatic drainage of Labia Minora glands</p> <p>* Lymphatic drainage of Labia Majora glands</p> | <p><u>Homologus</u></p> <p><u>Male</u></p> <p>Bulbourethral gland (Casper's gland)</p> <p>Prostate.</p> <p>Superficial Inguinal Lymph Node</p> <p>Deep Inguinal Lymph Nodes (Lymph Nodes of Cloquet)</p> <p>Deep Inguinal L.N.</p> <p>Superficial Inguinal L.N.</p> |
|--|---|

VESTIBULE

⇒ Anteriorly → Clitoris

(14)

Posteriorly → Fourchette

Lateral boundary ⇒ Harts line

⇒ Structures opening in vestibule ⇒ Urethral opening;
vaginal opening;
2 Bartholin's gland opening;

* Bartholin gland cyst ⇒ Pea Size;

Bartholin gland ⇒ Located b/w L. Minora & L. Majora groove
& @ the junction of Ant. 2/3rd & Post. 1/3rd;

- ↓
- ↓
- ↓
- Cuboidal epithelium
- duct travels Medially & forward; & opens b/w L. Minora & hymen (@ 5 o'clock; 7 o'clock).
- @ opening its squamous; while along whole length it is transitional epithelium.
- all kinds of cancer seen in Bartholin gland cancer
by "HONAN'S CRITERIA".

ii Asymptomatic Bartholin cyst $\oplus \Rightarrow$ No Rx*

ii Symptomatic / Recurrent cyst $\oplus \Rightarrow$ Marsupialization
↓

Extirpation of the duct to
prevent Recurrence.

ii ≥ 40 yr & have Bartholin Cyst $\oplus \Rightarrow$ Excision
↓
Tes Ca Risk*

Bartholin Abscess \Rightarrow i) E.Coli > Gonorroea;

ii) Gential Tx = I&D

↓

Marsupialization on Later date

* Secretion of Bartholin gland \Rightarrow Alkaline; $\xrightarrow{\text{Released during}} \text{coitus}$
bc Acidic environment is spermicidal.

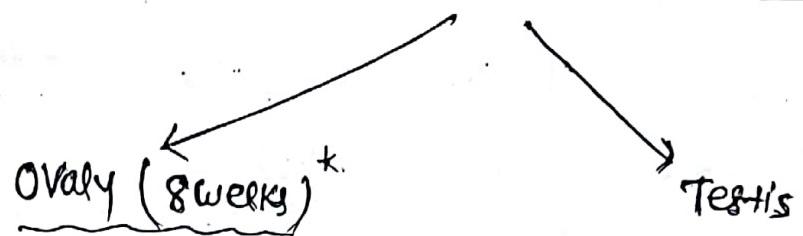
* they are content of Superficial Perineal Pouch;

RELEVANT EMBRYOLOGY

29

(15)

* Gonads \Rightarrow develop by Genital Ridge - 5 weeks^{QQ}



- Absence of Y chromosome

- Y chromosome

In XO genotype \Rightarrow ovaries appear
normally

- SRY functional part of

Y chromosome

@

6-7 weeks

distal segment

b/c "XX" genotype of short arm
Require for function. of Y-chrom.

ii) Nothing Mention;
differentiation b/w ♂ & ♀
② 12 weeks of P.O.G.

Streak Ovaries

↳ Seen in Turner Sy.*

* Gonads can be differentiated into ♂ & ♀ by 7 weeks

* Internal genitalia differentiates @ 10 weeks;

* External genitalia differentiates @ 12 weeks;

- Ovary part. in "Ovarian Fossa"

↳ part. In Lateral Pelvic wall

* Posterior to ovarian fossa \Rightarrow Ureter; Internal iliac vessels;

Anterior to ovarian fossa \Rightarrow Obliterated umbilical A.; Mesovarium

Lateral to ovarian fossa \Rightarrow Obturator Nerve & vessels

Pain from ovaries referred to Medical aspect
of thigh b/c of cutaneous br. of obturator Nerve,

Medial ⇒ Ovarian Ligament

Superior ⇒ External Iliac vessels.

Posterior ⇒ Levator Ani

Ovaries ⇒ 3 Supports

→ Ovarian Ligament Ovarian ligament

→ Infundibulum pelvis Ligament Infundibulum pelvis ligament
(Suspensory Ligament)

- Ovarian Vessels (gt called ovarian A. & veins)
- to Lateral Pelvic wall (Attaches ovary to Lateral Pelvic wall)
- cut - for oophorectomy

Mesovarium → False Ligament False Ligament

↳ Fold of Peritoneum

to posterior leaf of Broad Ligament,

OVARY ⇒ In Reproductive age Average 7-8 cc upto 20cc Normal
in Postmenopausal age average 2-3 cc upto 10cc Normal

Blood Supply ⇒ * Ovarian Artery ⇒ br of Abdominal Aorta
@ L2

Rt. ovarian vein

Ovarian vein ↳

Rt. drains into gvc

16

Lt. ovarian vein ↲ Lt. drains into Left Renal vein → gvc

Varicocele ⇒ M/c on left side; b/c ~~it makes 90°~~↳ M/c Reversible cause of Male Infertility(Lymphatic drainage ⇒ Para-aortic group of \wedge Lymph nodes)

Epithelium ⇒ Germinal epithelium

(single layer cuboidal)

10 Germ cells (Primordial Germ cell) ↳ Germ cells are formed here

↳ Epiblast (Ectoderm) ⇒ olden days from Yolk sacs

* Ovary has — Cortex — Follicles
— Medulla — Vascular

* Menopause ⇒ Follicles goes into programme cell death cause it

Epiblast ↳ Max^{num} follicles @ Intrauterine life 5th Month (20 weeks)
Yolk Sac (@ 3 weeks) ↳ 7 million

Genital Ridge (@ 6 weeks) ↳ On Birth = 2 million (1-2)

Oogonia (@ 9 weeks) ↳ On Puberty = 400,000

1^o oocytes (@ 12 weeks) ↳ Ovulate = 400 ovulate

Follicle formation begins (@ 14 weeks) ↳ 1000 atresia / Month

Follicle formation complete (@ 24 weeks)

Follicle / oocyte



Folliculogenesis

Folliculogenesis

↓
What is happening
in surrounding cells

Oogenesis

Oogenesis



What is happening
in Germ cells

- 1° Follicle - flattened

↓
Granulosa cell

1° Follicle - cuboidal

↓
Granulosa cell.

2° Follicle - Theca cells

↓
⊕

Antral Follicle - cavity ⊕

* all follicles have 1° oocyte @ centre; but ovum have
 2° oocyte.. * oogenesis begins IUL \Rightarrow @ 9 weeks



Oogonia

Mitosis

1° oocytes - diploid



Melosis I

(17)

Mitosis - I

Arrested Iⁿ \Rightarrow Prophase



diplotene

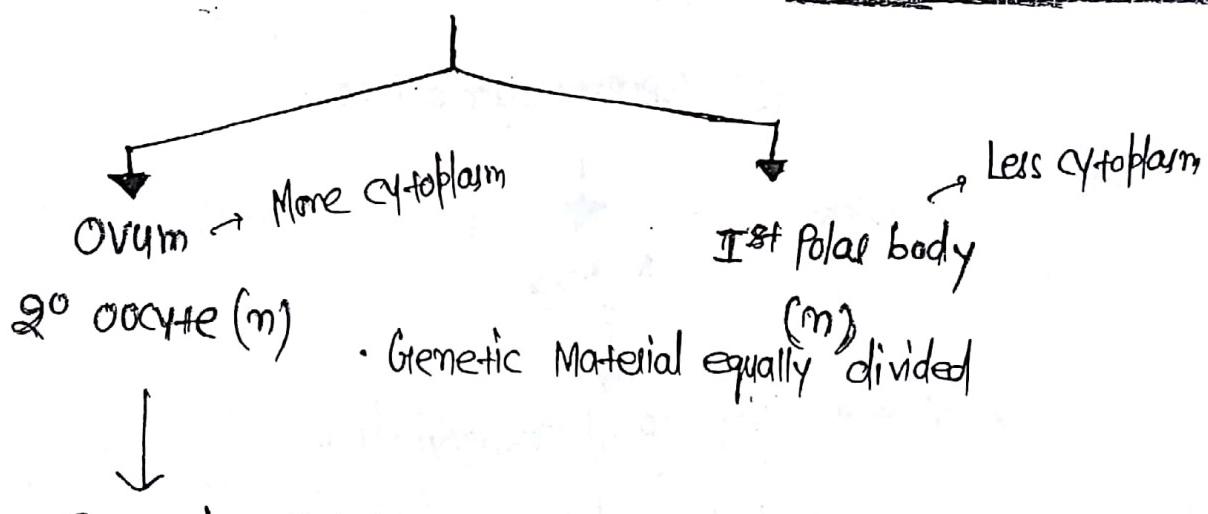


Arrested stage

* Diplotene

(Stage b/w Prophase & Metaphase)

When completed ?? - just before ovulation
 @ ovulation (3-4 hours before ovulation)



Enters into Mitosis - II

Arrested - II - Metaphase *

Completed - After fertilization

Female Pronucleus *

Ind Polar body



Fig. Fertilized ovum *

* Life span of ovum = 24 hr.

* Size of Mature ovum = 120 mm in diameter

Size of Mature follicle = 18-20 mm in diameter

Size @ which follicle Rupture = 18-20 mm *

SPERMATOGENESIS

- * Begins — Puberty in seminiferous tubules.
* duration — 72 day

① Spermatogonia ($2n$)



MITOSIS

(1) 1° Spermatocyte ($2n$)

Meiosis-I

Each

(Dictyotene absent)*



2° Spermatocyte

2° Spermatocyte



Meiosis-II

Spermatids

Spermatids

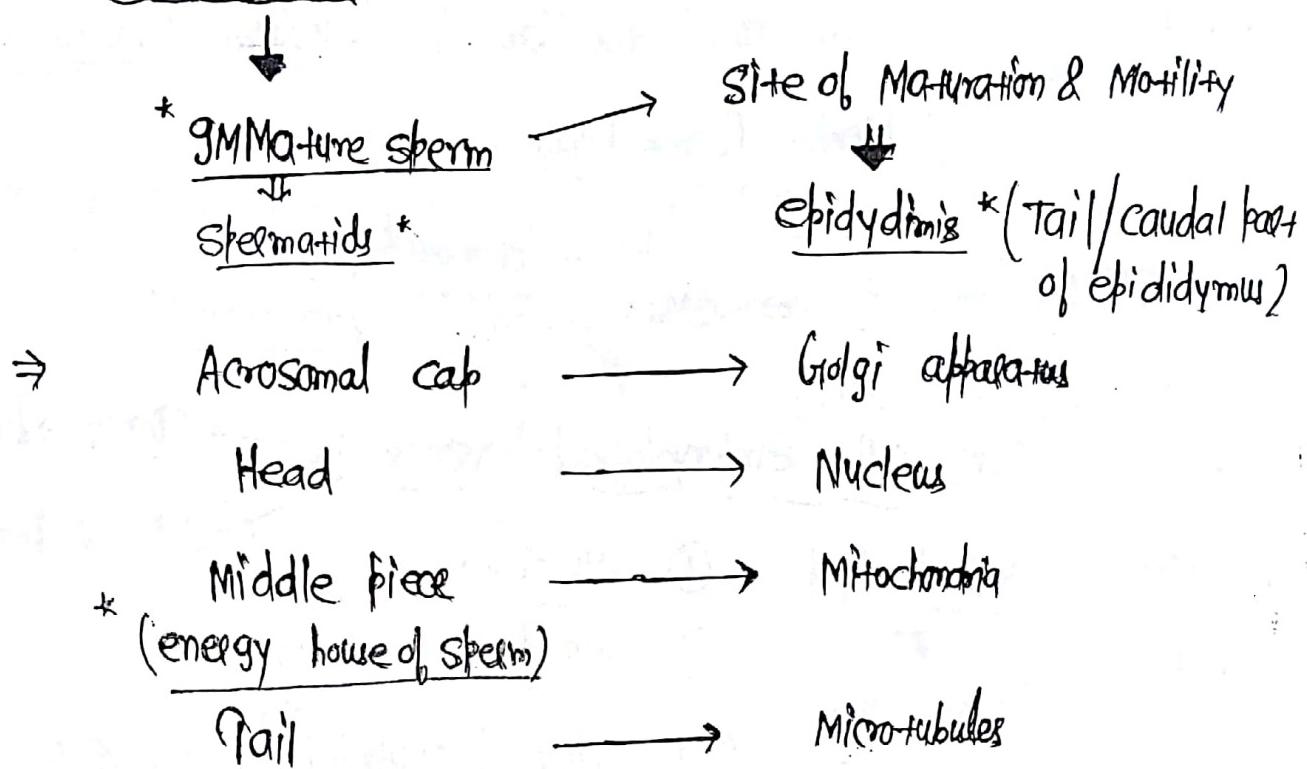
Meiosis-II

Spermatids

Spermatids

- * One 10 Spermatocytes gives \Rightarrow 4 spermatids.
- * one Spermatogonia gives \Rightarrow 16×10^6 Spermatocytes = 64 sperm
- * Fertilizable Life Span \Rightarrow 3 days; (18)
- * Mature sperm \Rightarrow ~~55 μm in length (smaller than ovum)~~
- * No. of sperms produced in one day = 100 million *
(Average sperm count)

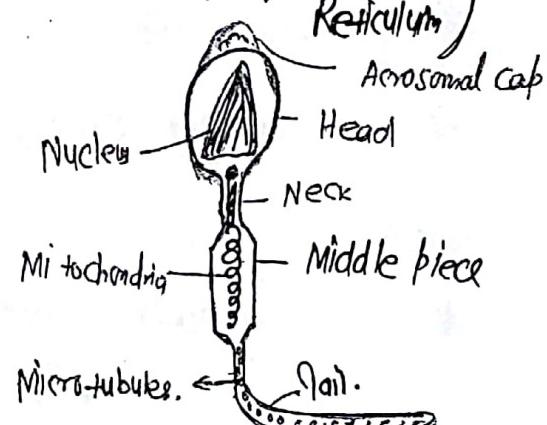
* Spermiogenesis \Rightarrow 12-14 days



. Sperms don't have ~~ER~~ Endoplasmic Reticulum (Rough endoplasmic Reticulum)

* Gene Responsible for Motility of Sperm \Rightarrow Calcium ~~ER~~

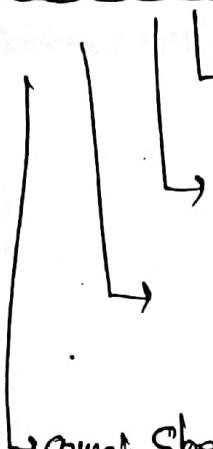
* Gene for Motility \Rightarrow CAMPSPER



FERTILIZATION

Capacitation

→ begins in cervix



Major site = Fallopian tube

Sperm = Reach the ampulla ∞ in 30 min

takes 7 hours.

lost just before implantation
on D₅
Prevent Polyfertilization
Receptor for sperm! ZPG

→ cause → Sperm take ability to bind zona pellucida

Sperm get the ability to undergo Acrosomal Rxn

Sperm React

Memb. Permeability — cat²

↳ hyper motility

* Acrosomal Rxn ⇒ Main Enzyme! Hyaluronidase

* For all Embryological events \Rightarrow Days \Rightarrow from Fertilization

- 3 Rules \Rightarrow
- ① 28 day;
 - ② 14th day — ovulation
 - ③ days of ovulation = days of Fertilization
- \Rightarrow Weeks \Rightarrow from LMP (1st day)

* 1st cleavage occurs \Rightarrow 20-30 hour after fertilization

* Conceptus enters in Uterus \Rightarrow Morula Stage @ 4 days
 \downarrow
16 cell stage; 8 cell stage

* Implantation occurs \Rightarrow in Blastocyst form
↳ on D₆ begins (D₆-D₇) ↳ on D₅ it form

(19)

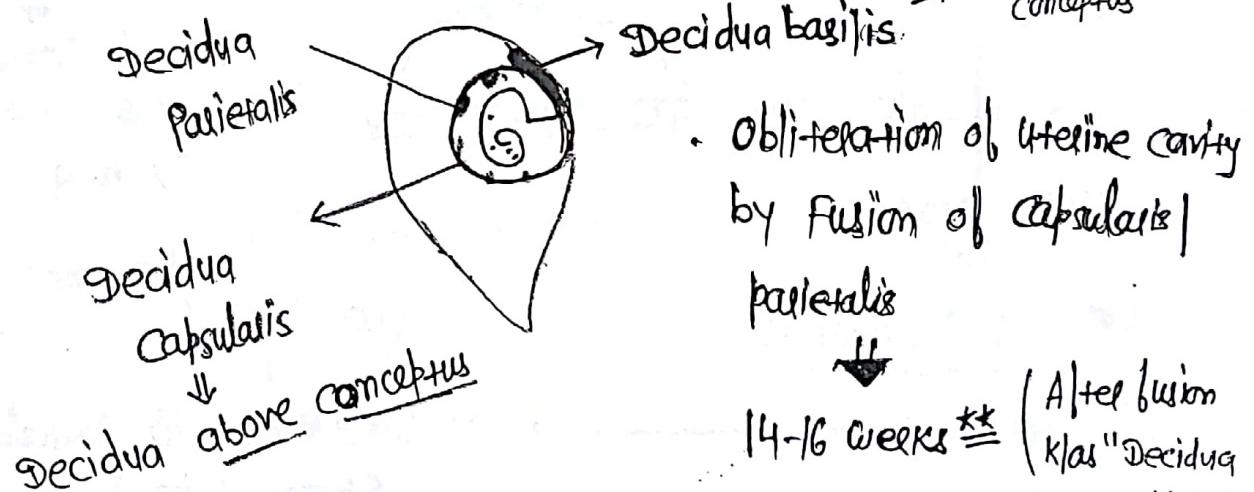
* Gestation occurs in 3 phases →

1. Apposition → Selecting
2. Adhesion → Inegrating
3. Invasion → Matrix Metalloproteinase

* Gestation completed @ D₁₀

* M/c site = upper posterior wall (Eccentric)
↳ one half bigger than other side

* Endometrium of ♀ → Klas "Decidua"
decidua below conceptus



- Obliteration of uterine cavity by fusion of capsularis | parietalis

↓
14-16 weeks ≈ (After fusion
Klas "Decidua vera")

SUPERFETATION

- Fertilization of 2 ova by 2 different sperms by 2 different acts of coitus

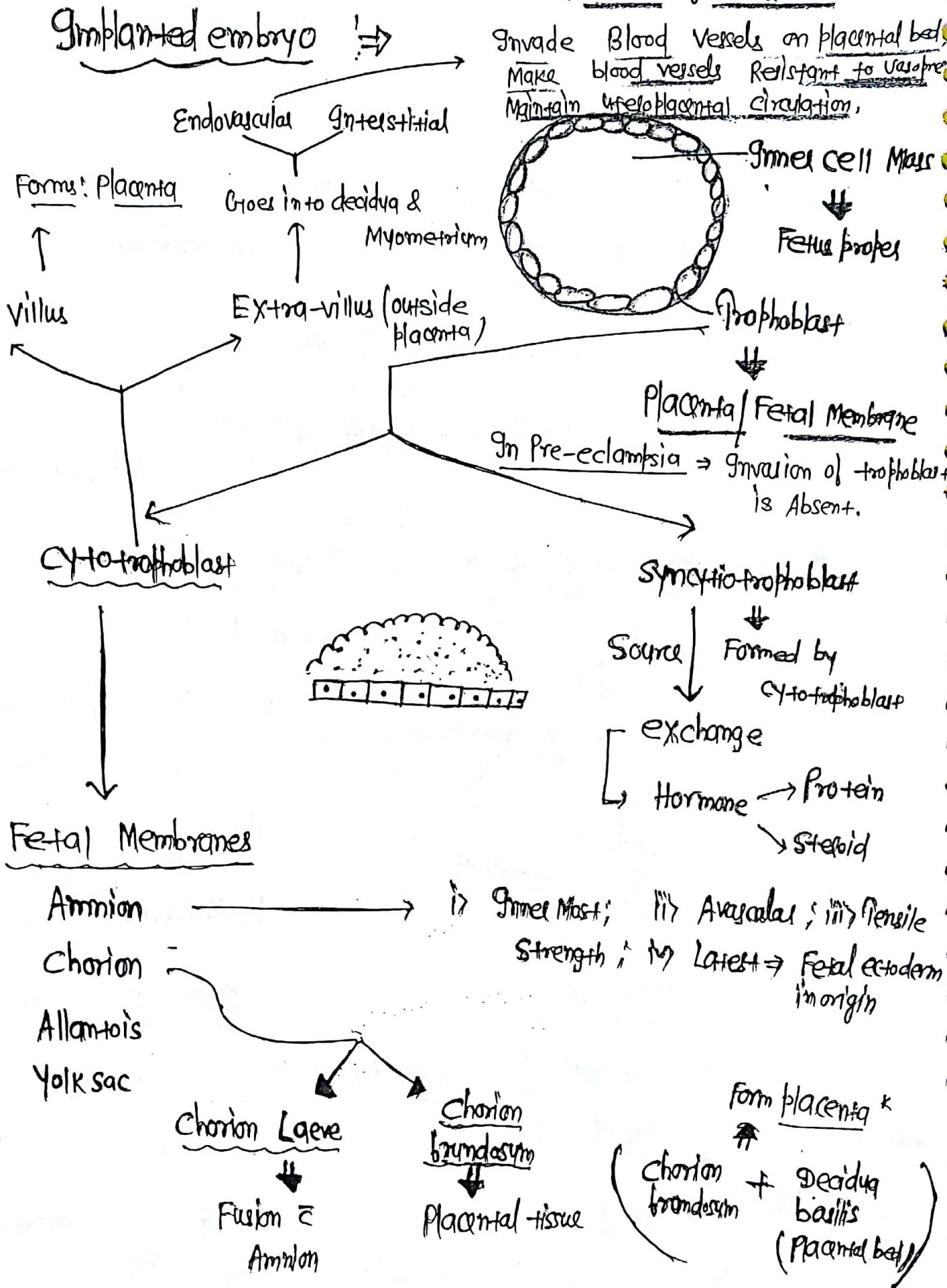
Different
↑
Menstrual
Cycles

No + documented
in Human

↑
2
Same
Menstrual cycles

- Theoretically twinning can happen upto 16 weeks.

* Embryonic phase lasts upto 8 weeks post fertilization & 10 weeks from L.M.P.



Allantois \Rightarrow Diverticulum from hindgut grows into connective stalk.

↳ Umbilical vessels (gt gives rise Umbilical vessels).

(20)

Yolk Sac \Rightarrow 1st site of hematopoiesis

↳ 3rd - 6 weeks (Portland, "Gottsch"; Graevene)

> 6 wks - Linear \div HbF \Rightarrow $\alpha_2 \beta_2$

> 20 wks - Long bones

- Bigger in size & shorter in life span.
- * Fetal Hb is resistant to both alkali & acid denaturation

↳ higher affinity for O_2
• Yes 2,3 DPG & carbonic anhydrase; Hb-O₂ curve shift to left.

ALKALI DENATURATION TEST

APTT (test)
 \downarrow

1. NaOH used

ACID DENATURATION TEST

- KB (Kleihauer Betke)
- Citric acid & Pgy buffer used.

Bed side test

- Qualitative test
- Maternal blood & Fetal blood from each other
- differentiates
- ↳ Maternal blood $\xrightarrow{\text{Moms} \Rightarrow \text{O}^{\text{ve}}$ (colour change)}
- ↳ Baby's $\xrightarrow{\text{Baby's} \Rightarrow \text{O}^{\text{ve}}$ (colour resistant)}
- Laboratory test
- Quantitative test
- Fetal RBC from Maternal RBC \rightarrow Count.
- Used in Rh^Ove ♀ to calculate dose of Anti-D.
- Singer's test (other kind of alkali test)

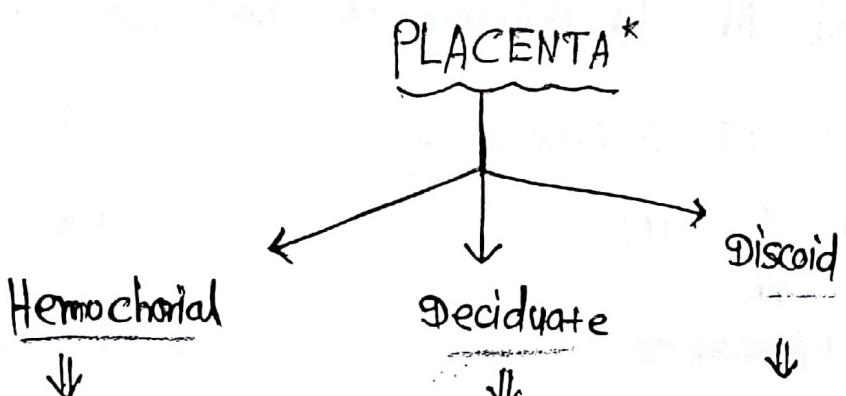
In Ante partum hemorrhage we can do "APTT test". *

* Fetal RBC → Larger in size
Shorter Life → 90 days *

- (Hb) of baby @ birth ⇒ 18 gm%.
↓

75-80% of HbF & Rest Adult Hemoglobin.

- @ 6 Month < 1% HbF part. out of total.



- Maternal & fetal blood doesn't mix.
- Sheds off • Shape

Wt of term placenta = 500 gm

volume of term placenta = 500mL (volume)

Diameter = 20cm

Thickness = 2.5cm

At term: Placenta: Fetal ratio = 1:6

Maternal side

Facing decidua

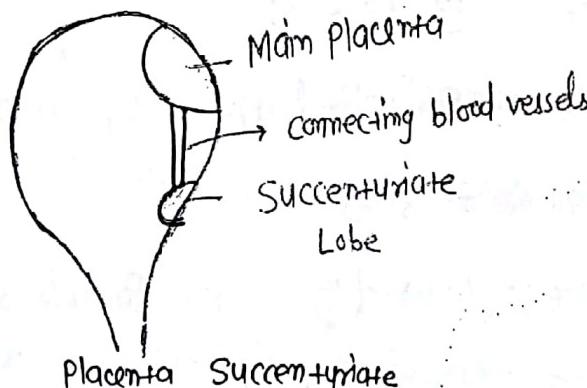
Lobes (Divided into 15-20 lobes). Identify \Rightarrow Smooth / shiny

\downarrow each lobe divided into

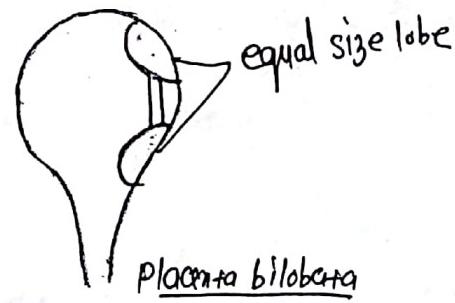
Lobules (Functional Unit) \downarrow 3-5 lobules
 \downarrow like "cotyledon!"

- * Placenta bilobata \Rightarrow Placenta is separated into lobes;
 - Division is incomplete & the vessels of fetal origin extends from one lobe to the other before uniting to form umbilical cord.

- * Placenta Succenturiate \Rightarrow having Accessory lobe; which is connected to the Main part of placenta by blood vessels.



Placenta Succenturiate

C.S. do \leftarrow Obstetric emergency \leftarrow Fetal side

(2)

Facing the fetus

Identify \Rightarrow Smooth / shiny
 \downarrow (b/c of Membrane)

Umbilical cord attached



@ centre of placental disc

\downarrow umb cord attached to the periphery \Rightarrow Battledore placenta

It can cause Post partum hemorrhage \approx

Villamentous Placenta \Rightarrow if fetal vessels travel outside the cord for some distance before reaching placenta (\therefore More chance to injury).

Vasa previa \Rightarrow vessels are travelling over the internal os.
 \rightarrow type of villamentous placenta;
 \rightarrow Rare type of APH (Fetal blood loss).



Result in severe fetal stress

* Intervillous Space \Rightarrow Maternal blood

Inside villi \Rightarrow Fetal blood*

1^o villous \Rightarrow  \rightarrow Solid - $\oplus \text{D}_3$

2^o villous \Rightarrow  \rightarrow Merodermal core - $\oplus \text{D}_{16}$

3^o villous \Rightarrow  \rightarrow Blood vessel 

* Fetal blood flow through placenta \Rightarrow 400 ml/min.

Fetal circulation Established @ D_{21}

Uteroplacental circulation = D_{12}

@ $\text{Perm} = 450-650 \text{ ml/min.}$

* Intervillous space \Rightarrow 140 ml blood

\downarrow PO_2 in Intervillous space \rightarrow 120 spiral arterioles \oplus Inside.

- 35-40 mm of Hg. O_2 Saturation = 65-75%

Low pressure = 10 mm of Hg \rightarrow Invade spiral A

K[as] "endovascular"
* Cytotrophoblast -

\downarrow
Permanent vascularization

\downarrow
Good Uteroplacental circulation

\downarrow
Vascular Remodelling

* Vascular Remodelling is controlled by decidual Natural killer cells.

(22)

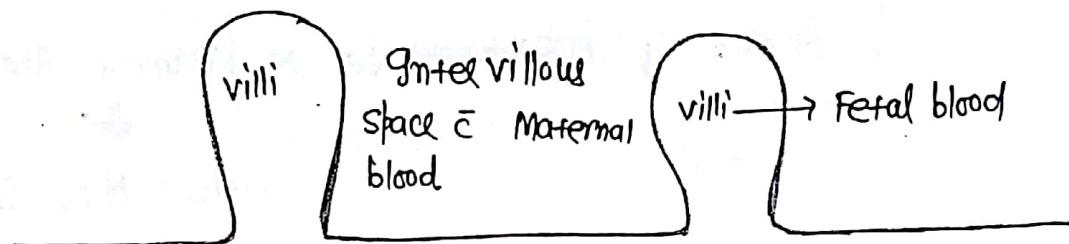
* Completed 2 phases

12 weeks

16 weeks

* Absent Vascular Remodelling \Rightarrow Pre-eclampsia
GUGR

* (AbN) Vascular Remodelling \Rightarrow Adherent Placenta*



PLACENTAL FUNCTIONING

• Placental formation begins @ 6 weeks.

Anatomically - Placenta completely formed by 16 weeks.

Physiologically - Maturation continues

- \uparrow POG \Rightarrow
 - Choriotrophoblast \downarrow
 - Syncytiotrophoblast Thickness \downarrow (thin)
 - \downarrow Stroma
 - \uparrow Fetal blood vessels
 - also keep moving towards periphery of villus
 - Hofbauer cells \oplus
 - \hookrightarrow Fetal Macrophages

term



No Choriotrophoblast
② term

Function of Placenta

→ Nutritional
Excretory
Respiratory
Endocrinol (Most imp)

Progesterone | Estrogen | HPL | HCG
↓
Human Placental Lactogen

Progesterone ⇒ Maintenance of ♀

↳ Smooth Muscle contractions
↳ If amount of progesterone ↓ ⇒ Recurrent Abortion
↓
Luteal Phase Defect (L.P.D.)

also do decidualization (Hypersecretory change)



On HPE ⇒ "Arias Stella Reaction"

↳ No Progesterone



No Arias Stella Reaction.

* Source of Progesterone in early ♀ ⇒ Corpus Luteum

Rescue the corpus luteum
from Luteolysis ←
↳ Pregnancy
↳ HCG
↳ Maintaining Corpus Luteum
↳ Natural late
↳ Luteolysis *

23

- * Placenta will take over the function of corpus luteum.



8 week (8-10 week)

Make Progesterone by precursor \Rightarrow Maternal LDL cholesterol

- * Corpus luteum of pregnancy will regress

\hookrightarrow 12 weeks

- * Ovarian cyst of 1st Trimester \Rightarrow May be enlarged corpus luteum
 \hookrightarrow Observation & Resolve after 12 weeks.

Estrogen \Rightarrow Specific to ♀ = E_3 Estriol.

E_2 = also formed; but Not specific

\hookrightarrow Growth of Uterus

\hookrightarrow Mask of ♀ = Melasma

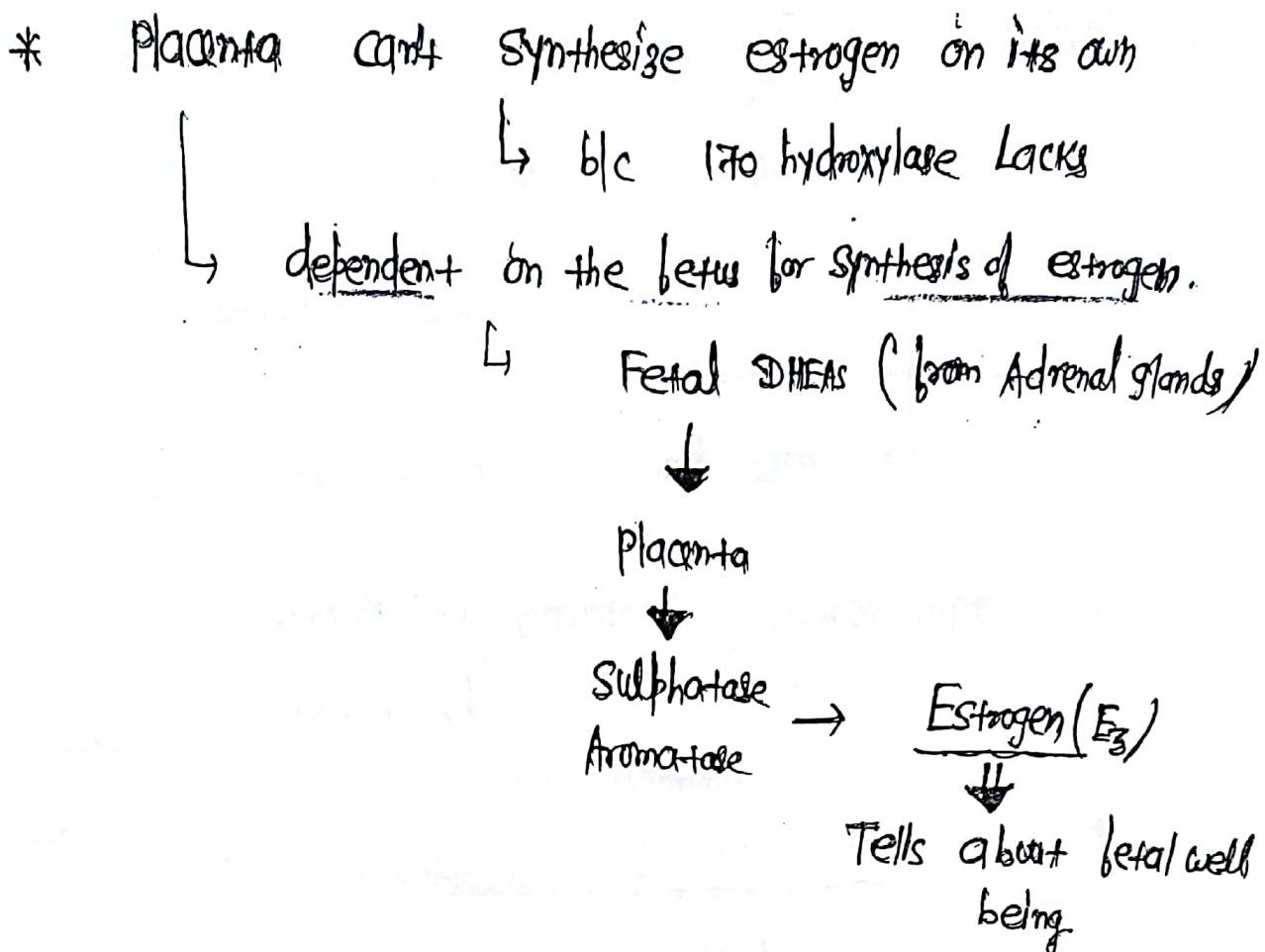
\downarrow
Hyperpigmentation

(Stimulates Melanocytes)

\hookrightarrow Retain salt/water

\hookrightarrow Obstetric cholestasis

\hookrightarrow Thyroxin binding globulin ↑



HPL (Human Placental Lactogen) | HCs (Human Chorionic Somato Mamotrophin)

→ GT tells about i) Placental functioning

↓

as P.O.G. rises \Rightarrow Matures \Rightarrow ↑HPL

Peak = 36 wks.

Q9 Which hormone is produced by placenta in maxⁿ amount,
at term = HPL (1gm/day).

i) endocrin function \Rightarrow Main function

↳ insulin resistance in ♀
(Plasma cortisol: growth hormone)

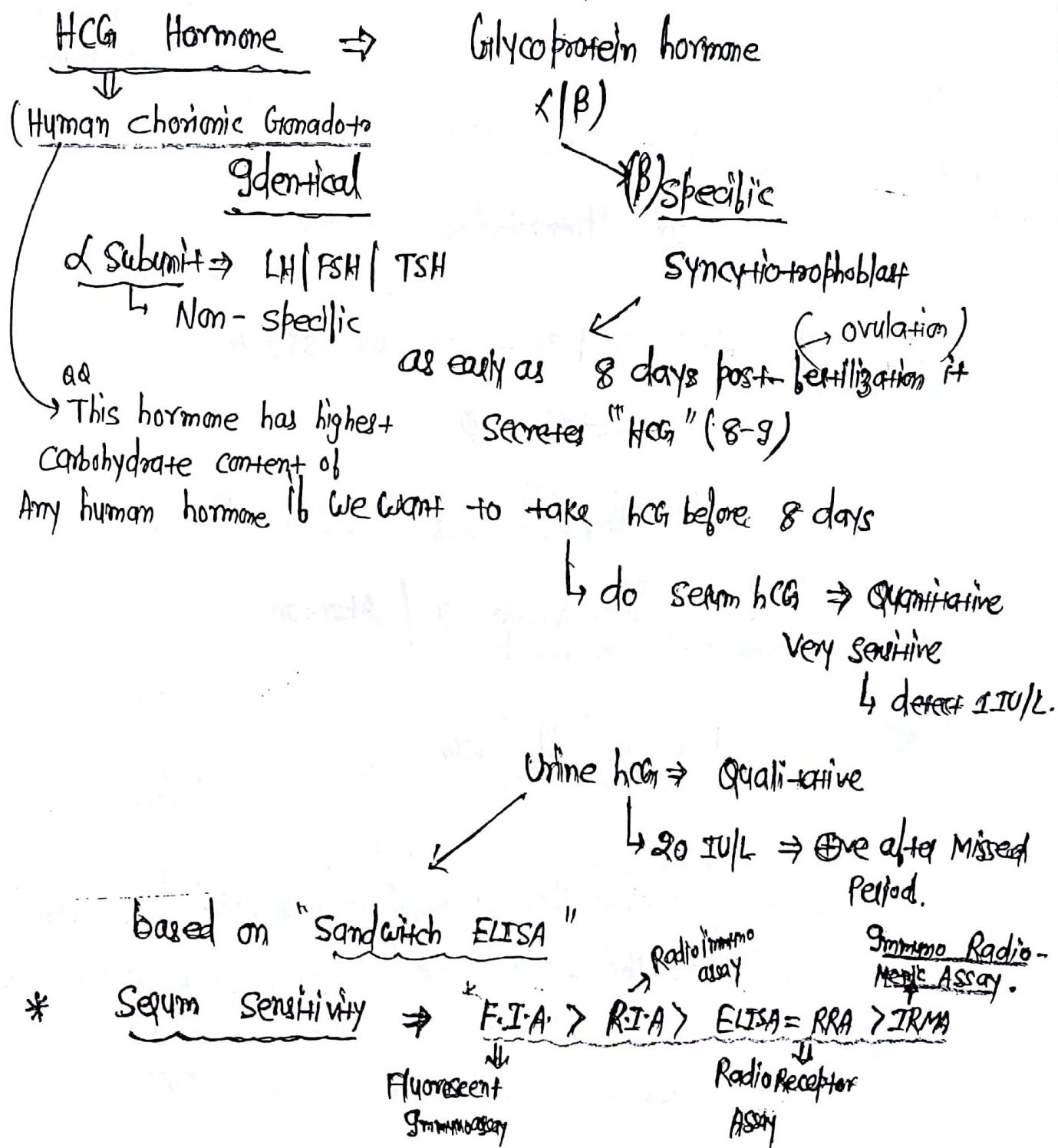
Q. Which hormone is responsible for fetal growth

↓

(24)

Insulin like growth factor / IGF

- * Promotes Maternal Lipolysis - Levels of free fatty acids are too which mother utilises as a source of energy; sparing glucose for fetus.



\Rightarrow hCG Value rises as P.O.H. rises

Max^m = @ 10 weeks*

→ normal pregnancy

After 10 weeks = rises

Doubling time of β -hCG

Min^m = @ 16 weeks

↓

After 16 weeks = plateau

48 hrs.

It means rise by 50% - 60%
Min^m after 48 hrs.

It does not mean 100%
or double

V.V.G.
Q9

1st \oplus_1 / \oplus_3 - rises by 55% ??

(N) Intrauterine Pregnancy

2nd \oplus_1 / \oplus_3 - rises by < 55% ??

Ectopic \ominus

3rd \oplus_1 / \oplus_3 - rises

Non-viable \ominus / Abortion

* Critical value of hCG for TVS = 2000 IU.

TAS = 6500 IU.

If hCG is More than or equal to these values & we don't see sac in the uterus \Rightarrow Likely to be ectopic \ominus .

* Cond'n where hch less than expected

- Multifetal ♀
- GTD (Gestational Trophoblastic Disease :)
- Down's syndrome
- Hyperemesis gravidarum
- Underestimated Gestational Age.

(25)

* Cond'n where hch less than expected

- Non-viable
- Ectopic ♀
- overestimated gestational age
- Trisomy 18.

- Functions of hCG \Rightarrow Maintenance of ♀ :

- ↓ Uterine contraction ;
- Growth & development of umbilical cord ;
- 1st stimulus for release of testosterone from Male foetus - hCG

• Immunosuppressant

QQ Why the conceptus Not Rejected ??

- i) Villous trophoblast Lacks HLA (MHC) ;
- ii) EVT (Extra villous trophoblast) - that have HLA-G1 \hookrightarrow only in Human skin Immunosuppressive

 decidua → NK cells have deficient cytotoxicity.

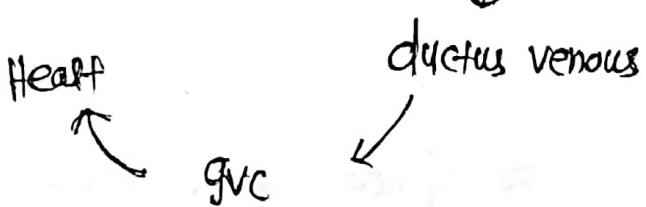
→ M/c insertion → center.

UMBILICAL CORD (Attached to fetal side of placenta).

- Average Length = 55cm (30-70cm)
- 3 vessels → 2 Umbilical A. (deoxygenated blood)
1 Umbilical vein (Left) "Right U. vein displace"

* Short cord < 30cm
* Long cord > 70cm

Umbilical from placenta
vein



* Max^m O₂ Saturation b/f. In ⇒ Umbilical vein
↓
80%.

* M/c Vascular Anomaly ⇒ SUA (2 vessel cord)
Ob. cord (Single Umbilical A.)

then check

GCA (Gross Congenital Anomaly)

↑ CVS,
↓

M/c gross
Anomalies

(Renal) ⇒ M/c Anomalies
Not gross ↓

↓ SUA

* If we see SUA + GCA

SUA + GCA

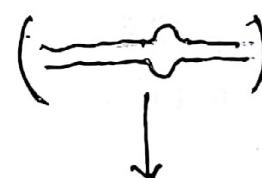
↑ Risk of Aneuploidy *

(26)

(Trisomy 18) **

* Isolated SUA → doesn't ↑ Risk of Aneuploidy *

* Incidence of SUA is higher in ⇒ Twin ♀ / Multifetal ♀
↓



↑ Risk of true knot of umbilical cord.

False knot ⇒ Protrusion of Wharton's jelly containing loop of umbilical vessels.
↓ No clinical significance

True knot ⇒ ↑ Risk of still birth;

- ↓ Cause ⇒ Fetal Movement.
- ↓ Active fetal Movements
- ↑ Risk in Twin.

1st Umbilical Artery



Umbilical vein



Ductus Venosus



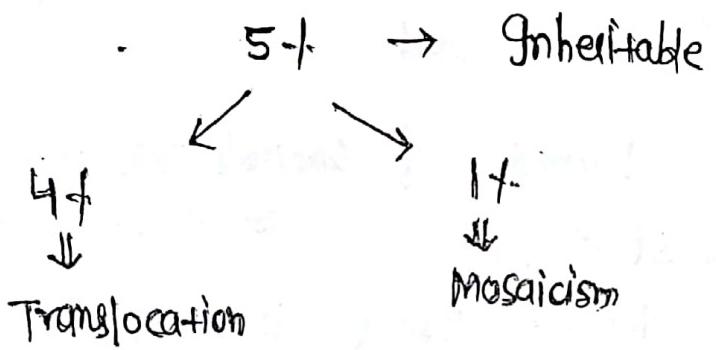
Ductus Arteriosus



Foramen Ovale

Down's SYNDROME *

- Trisomy 21.
- Causes \Rightarrow Most Common cause $\xrightarrow{*}$ Non-disjunction (95%)
 - during Meiosis-I
 - It is by chance; so;
 - Non-heritable in Nature.
- Recurrence = 1%.
- If 1st baby down's \rightarrow Antenatal testing done in Next ♀.



\approx 18+ baby \rightarrow Down \rightarrow (Translocation 21, 21)
↓
Risk = 100%.
 \hookrightarrow Abortion. (In 2nd baby)

Robertsonian Translocation seen in
Down Sx.

* All ♀ women

↳ offered down's screening. (opt-out Screening).

SCREENING1st Trimester

- $NT \geq 3\text{mm} \Rightarrow +ve \leftarrow$
- (Nuchal translucency)
- ↳ Fluid collection behind Neck;
- 11-13 weeks
- Dual test $\Rightarrow @ 11-13 \text{ weeks}$
- $\beta \text{ HCG} \Rightarrow +ve$
- PAPP-A $\Rightarrow +ve$

(Pregnancy associated plasma protein A)

* USG for NT + Dual test

↓
combined test*

Soft Markers \Rightarrow { Absent nasal bone (hypoplastic) \Rightarrow can be seen @ 1st trimester
Most imp. \Leftarrow { Nuchal fold thickness ↑ed,
↳ ($> 6\text{mm}$) \rightarrow Screening +ve

echogenic cardiac foci
echogenic bowel foci

- Short femur
- Short humerus
- Short frontal lobe
- Short ear length
- Simian crease (single palmar crease)
- Short 4th middle phalanx (climadactyly)
- Saddle gap
- Pyelectasis (mild dilatation of renal pelvis)

2nd Trimester (27)

SOFT MARKERS (Not specific)
 ≥ 2 Soft Markers

↑ Risk of Aneuploidy
15-20 weeks 99

Maternal Serum Marker

Triple test

↳ $\frac{\text{hCG}}{\text{AFP}}$
15-22 wks.

UE_3 (Unconjugated Estriol)

→ Quadruple test

↳ $\frac{\text{hCG}}{\text{AFP}}$
 UE_3

+ inhibin A Yes

↳ Produced by placenta during ♀ & corpus luteum in Non-pregnant female

QQ M/c Congenital cardiac Abnormality In Down's sx child ↴

Endocardial cushion defect > VSD > ASD

QQ Gastrochisis is Not Seen In Down's sx patient ↴
Not central (More towards one side)

Omphalocele ⇒ Covering Membrane (+)
↳ central

* Confirmatory test ⇒ Karyotyping
M/c Method : "G Banding"
In Metaphase
Drug: colchicine

1st Trimester
↓
Chorionic villous Sampling
⇒ 10 weeks
M/c ⇒ 11-13 weeks

don't do before 9 weeks

↓
b/c it may cause

Limb defects

- can result in false +ve test (placental syncytiotrophoblast)
- Risky fetal loss 1/4
- early result + trophoblast (48-72 hr)

2nd trimester
↓
Amniocentesis
≥ 15 weeks

M/c = 16-18 weeks

don't do 11-14 weeks

↓
Early amniocentesis

↓ Fetal loss

- More accurate
- More safe less sy.

• delayed (7-10 days)
Result ↓

Amniocytes
fibroblast

* Cordocentesis → Umbilical cord blood cells

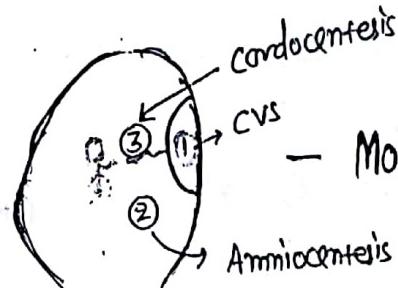
↓

(28)

from umbilical vein (placenta end)

↓

b/c it is stable
end.



- Most Risky ⇒ 3% fetal Loss.

* Amniocentesis: all USG guided procedure; Not blind - procedure.

* NIPIT ^{costly} (Non-Invasive Pre-Natal test) : ⇒

- Genetic Material ⇒ cell free fetal DNA
- done on Maternal blood (Some amount of cell free DNA circulating in Maternal blood)
- ≥ 10 weeks
- this test is used as 2^o screening (costly)
- has to be followed by confirmatory test;
- takes 7-10 days for Results.
- tells us about Trisomy 13, 18, 21
Monosomy - XO

PHYSIOLOGICAL CHANGES AFTER PREGNANCY

Presumptive sign

↓
Women experience

Reflux (eg → Amenorrhoea;
Morning sickness
etc)

Probable sign

↓
Seen by doctors

Positive sign

↓
Confirmatory

earliest = hCG ^{serum}
dx ^{urine}

USG

O/E → Fetal parts

↳ Fetal heart Rate

Probable sign ⇒ i) Hegar's sign ⇒ Softening of Isthmus

(on Bi Manual examination)

↳ Seen on 6th week

ii) Good sign ⇒ Softening of cervix

↳ Seen on 6th week

iii) Chadwick's (Jaquemier's sign) ⇒

Bluish discolouration of vagina / vulva

④ @ 8th weeks

iv) Osiander's sign ⇒ Lateral vaginal fornix pulsations

v) Piskacek's sign ⇒ one half of uterus feels more enlarged (Asymmetrical)

VII) Palmer's sign \Rightarrow Regular Rhythmic Uterine contractions
 @ 8th week (No Palp; only felt by examinee) (29)

VIII) Haltmann's sign \Rightarrow Implantation bleeding (Not a problem)
 @ 8th week

On USG \Rightarrow 1st sign of pregnancy on USG *

Gestational Sac **

On TVS \Rightarrow 4-5 weeks (4-3 day)
 empty bladder \rightarrow On TAS \Rightarrow 5 weeks
 Full bladder \rightarrow

TVS is ∞ higher frequency &
 $(\geq 5 \text{ Hz})$
 TAS is ∞ lower frequency
 (3 Hz) .

Intradecidual sign \Rightarrow Marker of intra-uterine ♀ early

Yolk Sac appearance \Rightarrow TVS = 5-5 week

TAS = 6 week

Fetal pole \Rightarrow Cardiac activity \rightarrow TVS \Rightarrow 5-6 weeks
 \rightarrow TAS \Rightarrow 6-7 weeks

* Double-decidua sign \Rightarrow Marker of early intra-uterine ♀.
 Decidua capsularis \nwarrow Decidua parietalis

* Double bleb sign \Rightarrow sign of early intrauterine
 ↙ ↘
 Amniotic Yolk sac ♀.

Q9. earliest to see Gestational Sac \Rightarrow 5th week
 on USG

Q10. Earliest time Gestational Sac can be identified on TVS
 from LMP ??

Q11. Earliest time Gestational Sac can be identified on TVS
 from fertilization ??
 ↳ LMP - 15 days = 30 - 15 = 15 days

\Rightarrow What ever seen on TAS can be seen in TVS (vice versa is Not true)

\Rightarrow Gn-Ectopic : Ring in Uterus \Rightarrow single Ring in Uterus

\Downarrow
 "Pseudosac" \Rightarrow present in ectopic uterus.

General Changes \Rightarrow ① Additional calorie Requirement

gn ♀; everywhere Progesterone
 except \Rightarrow Obstetric cholestasis $\begin{cases} \uparrow \text{estrogen} \\ \uparrow \text{salt & water retention} \end{cases}$
 ↑

\Downarrow
 350 Kcal/day
 \Rightarrow gn 1st trimester = No additional
 calorie requirement

Brought about by
 ↑
 Retain salt/water

② Average wt gain

\Downarrow 12 kg (10-14 kg)

③ BMR \Rightarrow ↑ by 20%

④ Salt/water \Rightarrow 6.5 L

(4) Plasma osmolarity \rightarrow Yes (10 mosm/kg)

(30)

(5) Plasma volume \rightarrow $\uparrow 40\%$

Red cell Mass \rightarrow $\uparrow 20\%$

Hemodilution condition \Rightarrow Anemia of Pregnancy

Anemia \Rightarrow Hb $< 11 \text{ gm}/\text{dL}$; Hematocrit $< 33\%$

\hookrightarrow M/C = Iron deficiency Anemia*

Total Fe Requirement = 1000 mg

300 mg (Fetus)

Best test = S Ferritin \rightarrow In fetus form

Prophylaxis = IFA (100 mg Fe + 500 µg FA)

\hookrightarrow 1 tab

\rightarrow 6 month during pregnancy

6 month after delivery

Treatment of Anemia \Rightarrow IFA tab (2 tabs) \rightarrow Acute blood loss & Hb $< 6 \text{ gm}/\text{dL}$

Parenteral

Blood transfusion

i) If the patient is Not compliant by oral tab

i) If $> 34 \text{ weeks}$ & Hb $< 7 \text{ gm}/\text{dL}$

ii) Not tolerating oral tab

ii) Even No sign/symptom of heart failure & Hb $< 5 \text{ gm}/\text{dL}$

iii) Malabsorption sx.

iii) Anytime sign & symptom of CHF

⑥ TLC \Rightarrow Tes (It doesn't Mean Infection)
↳ 15,000 during Primed.
25,000 after Postpartum.

⑦ DLC \Rightarrow Neutrophilia
ESR ↑
CRP ↑

⑧ Platelet count \Rightarrow Average platelet count Tes
↳ Not Causes Thrombocytopenia

⑨ Clofing factors \Rightarrow all Tes except \Rightarrow Factor II & B.

⑩ Insulin Resistance \Rightarrow Hyperinsulinemia
↑as POG Tes
Significant $>$ 240 mg



Fasting - Hypoglycemia

Post-prandial - Hyperglycemia

* Anemia : $< 11 \text{ gm/dl}$

Severe Anemia : $< 7 \text{ gm/dl}$

Very Severe Anemia : $< 4 \text{ gm/dl}$

Systemic

CVS **

(31)

↳ Plasma Volume $\Rightarrow \uparrow 40\%$

Red cell Mass $\Rightarrow \uparrow 20\%$

Cardiac output $\Rightarrow \uparrow 40\%$

$\text{O}_2 \text{ demand of tissue} = \uparrow 20\%$

feature of $\leftarrow \text{O}_2 \text{ carrying capacity} = \downarrow \text{res}^*$

Hb; Not of $A-V \text{ O}_2 \text{ gradient of tissue} = \downarrow \text{res}$

Red cell Mass

- All heart sounds are loud = Loud S₁
- S₃ (Galloping Rhythm)
- Systolic Murmur (Ejection systolic Murmur)
 - ↳ Physiological up to grade 2.

Diastolic Murmur

↳ almost/ Always Pathological

- Heart Rate Res (by 16-18 beats/m above baseline) (< 100)
 - Split S₁
 - Apex beat \Rightarrow heard @ 4th ges (b/c heart is rotated Anteriorly & Pushed up).
- on CXR \Rightarrow cardiac silhouette (appears big)
- cardiomegaly \Rightarrow always pathological

on ECG \Rightarrow LAD \Rightarrow Physiological
↳ Left Axis deviation**

- Blood pressure = SBP > SBP \downarrow (Both Fall)
(10 mm Hg) (all vaso pressure \downarrow)

E | P (Estrogen ↑ Progesterone)

- \rightarrow i) Vasodilation
ii) Resistance against vasoconstrictors

beginning - 5 weeks (\downarrow in SBP begins @ 5 weeks)

Maxim = 24-26 weeks

after 26 weeks = beginning \uparrow
(Come back to prepregnancy value)

* ≥ 20 weeks \Rightarrow Sublime hypotension syndrome

\downarrow
Gravid uterus compresses IVC

Changes position to Left Lateral Position

↳ ↑ utero-placental circulation

Fetal O₂ saturation by lox

* Preload \Rightarrow ↑

Afterload \Rightarrow ↓ (↓ fall in Systemic Vascular Resistance)

* Ejection fraction = No change

- Central venous pressure = No change

(32)

Persistently distended Neck veins



Pathological Alloys *

- CHF (Highest Risk)



Cardiac output Tes Seen

(Immediate Post-partum)
(3rd stage)

> 2nd stage of Labour > 32 weeks



CO is Not Tes until
it goes to 2nd stage of
Labour, after 32 week of
P.O.G.

~~Q~~ CO is Maxm @ ↴

a) 28 week; ~~b) 32 weeks~~; c) @ term; d) 36 weeks

~~Q~~ Pre-eclampsia Not commonly dx in

↳ 3rd trimester.

~~26/4/18~~

KIDNEY

⇒ Tes Renal blood flow by 80%.

GFR tes by 50%.

⇒ S Creatinine / BUN ↑;

S Urine acid Level \Rightarrow No change b/c Reabsorption.

* S. Creatinine ↑ & Pre-eclampsia*

S. Uric acid

* Kidney - enlarge by 1cm

* Hydrometer - b/c of Progesterone

↳ Smooth Muscle Relaxant

↳ Rt side > Lt side; Why??

In ♀ Uterus becomes dextro Rotation towards Rt side

↳ compresses Right Ureter

@ the Pelvic Brim.

but still doesn't become non-functional
as urethral pressure also ↑

↳ Bladder Pressure Test

↳ From 8cm H₂O

↳ To 20cm H₂O.

* Urinary Stasis ↳

Urine → Routine - Asymptomatic Bacturia $> 10^5$

→ Microscopic

↓

Treat; if we don't treat then
chances of Pyelonephritis becomes high.

* Glycosuria - b/c Renal threshold rises.

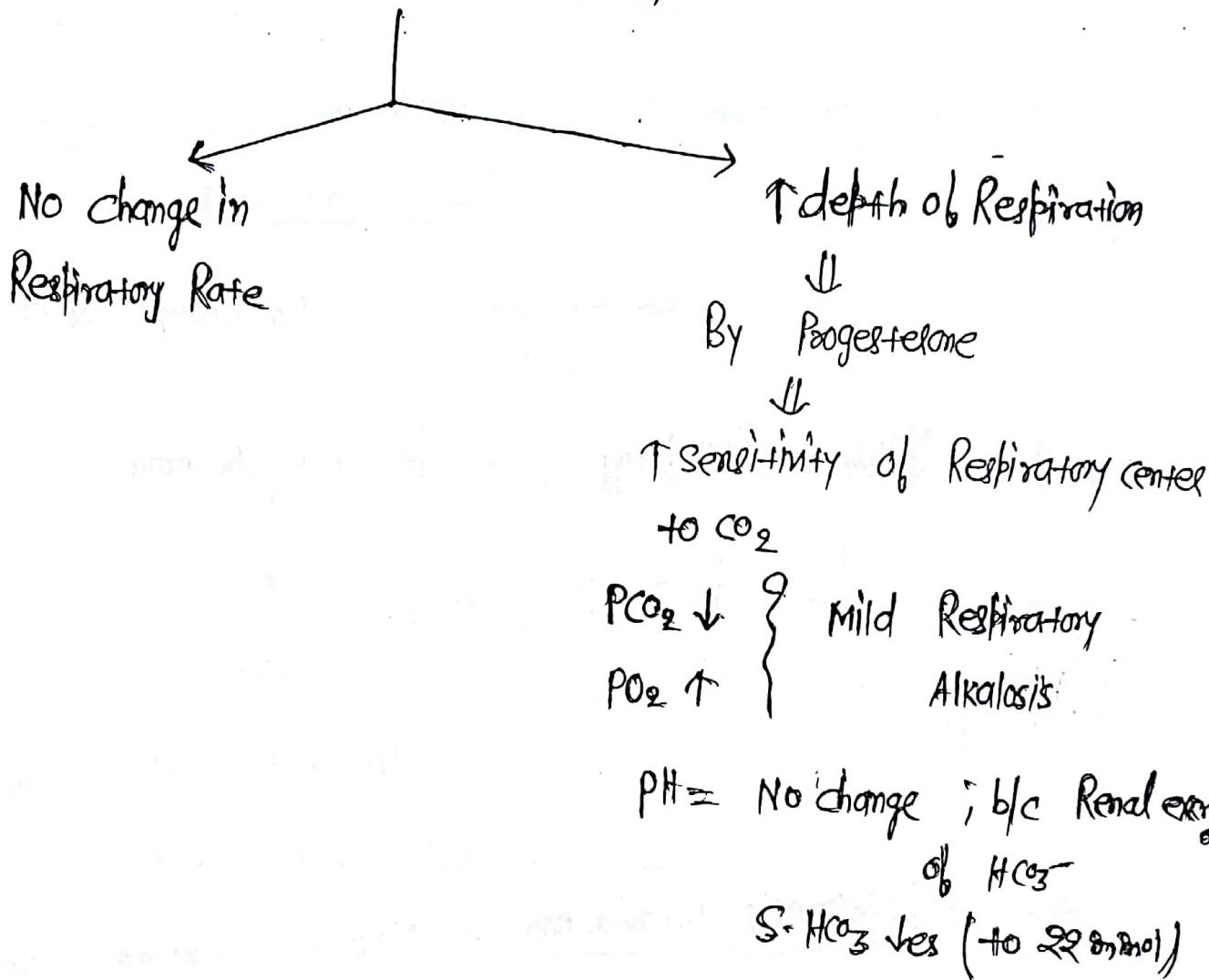
* Proteinuria - $\geq 0.3 \text{ gm/24 hr}$ of urine sample

↳ Not Physiological; it is Pathological

Respiratory System

(33)

- * Tidal volume Yes
- * Minute ventilation ↑ (40%)



- * Diaphragm rises by 4cm

↑ transverse thoracic diameter = 9cm

Total Lung capacity = Yes

Vital capacity = No change

so, ERV ↓ Yes ; IVC = No change = RR (Respiratory Rate)
 RV ↓ Yes

pH
 ↓ Fev,
 ↓ PCWP

* Diaphragmatic Excursions \Rightarrow Respiratory Function in Pregnancy:

GITP

- * GERD Seen \Rightarrow b/c LES tone less by Progesterone & Gastric Pressure less.
- * Gastric emptying time shows No change in all trimesters.
- * Nausea, Vomiting = d/t hCG hormone
 - \hookrightarrow 1st Line drug in ♀ = Pyridoxine
 - \downarrow No Response
 - Doxylamine + Pyridoxine
- * Hypertemesis Gravidarum \Rightarrow Nausea & vomiting causes either
 - $\geq 5\%$ cut. loss
 - &/or
 - Ketonaemia
 - \hookrightarrow d/t hCG
 - \hookrightarrow Infection of H. Pylori

Seen in Multifetal ♀ ; & female sex fetus;
- * ♀ vit. deficiency \rightarrow vit.K - Coagulation defect
 \rightarrow vit.B₁ - Wernicke's encephalopathy,

- Can cause Esophageal tear (Mallory-weiss tear);
- Renal failure (Acute Renal failure). (34)

\rightarrow Stop all oral intake
give i/v fluids + i/v Antiemetics.

- * Liver \rightarrow
 - ALP = Gross Tes
 - b/c Placental - ALP
 - Not a Marker of cholestatics (it is physiological tes)
 - Production of Albumin/Globulin
 - S. proteins \rightarrow Fall (b/c Production is less than Volume Tes)
 - AST; ALT both tes

Endocrine System*

Pituitary \rightarrow N In size 135x

b/c Lactotroph (Vascular supply T)

9m Sellar PPH — Vascular Supply — \downarrow to Pituitary

\downarrow
Infection

"Sheehan's syndrome"

Sheehan's Sx \Rightarrow M/c Presentation \Rightarrow Failure to Lactate

2nd M/c \Rightarrow Amenorrhoea

Usually - Ant. Pituitary affected



Post. Pituitary spared.

\Rightarrow If a ♀ doesn't Lactate - Menses by 6-8 weeks.

S. Prolactin Level - Higher

(Pregnancy)

Lactation



After delivery S. Prolactin level by 50%

Prolactin - Milk Synthesizing hormone

Oxytocin - Milk Let down / Ejection hormone

1st stimulus \Rightarrow Initiation of Lactation
 $(\downarrow E + P)$

Fall of E & P also causes \Rightarrow Post-Partum depression (Blues)

- THYROID \Rightarrow
- Thyroid binding globulin \uparrow (Estrogen) \downarrow (35)
 - Total T_3 & $T_4 \uparrow$ } $\Rightarrow \uparrow$ Production from the gland
 - Free T_3 $T_4 \uparrow$ (slightly) \downarrow
 - TSH \downarrow (slightly) Why? \Rightarrow b/c of hCG
 - I_2 requirement \uparrow \downarrow $\alpha = TSH$ (some)
 - (♀ & Lactation) both have RDA = 250 mg/day, as TSH,
- Pregnancy is Euthyroid condition
- I_2 excretion test.

- * M/c of hypothyroidism in $\text{♀} \Rightarrow$ Hashimoto's ds
 M/c of hyperthyroidism in $\text{♀} \Rightarrow$ Graves ds.

Q K/c/o hypo-thyroidism ; L-thyroxine 25 mg

Dx = Pregnancy \Rightarrow TSH \uparrow by 50% (b/c some part of thyroid doses become degenerated)
 Hypothyroidism May cause abortion.

Maternal Nerve Injury \Rightarrow M/c in Lithotomy position

Common Femoral Nerve

* M/c in Postpartum / Peripartum / Intrapartum /
 \downarrow
Lateral cutaneous N. of thigh $>$ Femoral N. \downarrow Extended Lithotomy position

* Foot drop in ♀ is diff "Lumbosacral plexus compression."

* Fetal Swallowing \Rightarrow 10 weeks

Fetal breathing Movement \Rightarrow 11 weeks

Fetal Urine production \Rightarrow 12 weeks

Fetal Meconium production \Rightarrow 16 weeks

IgM production in baby \Rightarrow 20 weeks

by Mother \leftarrow IgG transfer in baby \Rightarrow 16 weeks

Surfactant synthesis begins \Rightarrow 20 weeks

Surfactant appears in Amniotic Fluid \Rightarrow 28 weeks

Gluca^{gen} production \Rightarrow 8 weeks

Grenulin production \Rightarrow 12 weeks

H-P (circulation) \Rightarrow 12 weeks*

AMNIOTIC FLUID

(36)

Major Source \Rightarrow Fetal Urine

Major Removal \Rightarrow Fetal Swallowing

Major Source \rightarrow In 1st 12 weeks \Rightarrow Ultrafiltrate of Maternal Plasma
 In 12 - 20 weeks \Rightarrow Transudate across fetal skin
 In > 20 weeks \Rightarrow Urine.

* Amniotic fluid \Rightarrow Water (Nutrition)

\hookrightarrow Not help in Nutrition.

* Colour of Amniotic fluid \Rightarrow Straw coloured

Green colour = Meconium-Stained

Dark = Abruptio

Golden = Rh Incompatibility

Tobacco Juice (Dark brown) = GUD

Greenish Yellow (Saffron) = Post Maturity

* pH = 7-7.5

* Osmolality = 260 mosm/l.

* Water is Replaced in every 3 hrs.

Normal

AFI (Amniotic Fluid Index)
5-24 cm

DVP (Deep Vertical Pocket)
2-8 cm

Polyhydramnios

≥ 25

≥ 8

Oligohydramnios

≤ 5

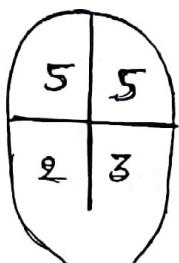
≤ 2

More Common

Method

Bettie (No quadrant
So; Less
error)

AFI \rightarrow



\Rightarrow divide in 4 quadrants
Arbitrarily

1S = Amniotic fluid.

DVP \rightarrow Not divided in quadrant; only pocket division.

* Absolute value \Rightarrow Polyhydramnios = 2L

Oligohydramnios \equiv 200 mL

* In twin ♀; We can't use AFI ; only use DVP;

b/c we don't know about quadrant of both Fetus.

(37)

Mild → Poly → Pathological
 → oligo.

Moderate - Severe → Poly → Gross congenital Anomaly
 → oligo.

GIV > NTD

Leakage of CSF in Amniotic Fluid

B/L of Fetal
Swallowing Problem

duodenal Atresia

double bubble sign.

B/L Renal Agenesis

Posterior Urethral valve

"Key hole sign" on USG seen.



Lady comes to Poly / oligo (Moderate - severe)

diabetes

Leaking Amniotic fluid

seen by Per speculum examination.

* Polyhydramnios have high Risk of → (Moderate - severe)

- ① Maternal Respiratory distress;
- ② Pre-term Labour - < 37 weeks
- ③ Premature Rupture of Membrane — Membrane Rupture before Labour.

Pre-term premature Rupture of Membrane - > 37 weeks & PROM.

④ Abruptio — Separation of Placenta from
Underlying decidua,

↳ prevented by Controlled ARM

↓
Multiple pin prick holes onto the
Membrane

⑤ Cord prolapse

↳ Obstetric emergency b/c of temperature
Change to → vasospasm
cord

↳ Severe fetal distress

do emergency C.S.

- i) Reposit the cord above the presenting part;
- ii) Fill the bladder;
- iii) elevate the buttock;
- iv) O2 by mask or iv fluids given.

* We do emergency CS; depends on

(38)

↳ { Cord pulsation (Absent)
Fetal heart Rate (Absent)
 do vaginal delivery.
 (GUD case)

(6) Malpresentation;

(7) PPH;

(8) GICA;

(9) Diabetes;

(10) Amniotic fluid embolism

* Oligohydramnios have high Risk of ↳

(1) Pulmonary hypoplasia;

(2) Fetal distress (cord compression)

(3) Malpresentation

(4) GUGR

(5) PE (Pre-e)

(6) Early in pregnancy oligo.

↳ association w oligo. b/c of
Heteroplacental Insufficiency (UPI)

↳ viral infection (TORCH/zika)

⑦

Amniotic band Sequence \Rightarrow

tears in Amniotic Membrane



Serous oligohydramnios (dI/f leakage.)



Bands & tight wrap around fetus.

M/c Anomaly in Amniotic band sequence



Limb Anomalies $>$ Craniofacial Anomalies

⑧

Ammion Nodosum



S. oligo + yellowish Nodules, on the Membrane
↑ serous

⑨

compression defect \Rightarrow CTEV (club foot)

Q&A

Diagnostic Amniocentesis \Rightarrow ① Karyotyping,

② Neural tube defect \rightarrow AchE/ AFP *

Best Screening test for NTD \Rightarrow USG

Best test for NTD \Rightarrow AchE/ AFP (Amniocentesis)

Other Screening test for NTD \Rightarrow Serum Alpha-fetoprotein

16-18 wks

$> 9 - 10 \text{ times} \text{ normal}$ measured median,

AFP Peak → In Fetus - 13 weeks

 In Mother - 39 weeks

 $t_{1/2} = 5-7 \text{ days.}$

39

* ↓ Level of AFP seen in \Rightarrow Down's Sx;
Diabetes;
Obesity;
Molar \oplus ;
GUD.

③ Lung Maturity $\Rightarrow \text{M/C} = \text{L/S Ratio} \Rightarrow > 2 \Rightarrow \text{Mature}$

↳ Best test $\Rightarrow \text{PGI} (\text{Phosphatidyl Glycerol})$

↳ Not affected by presence of contaminants.

→ in Diabetes Mother.

⑨ Hemolytic Anemia \Rightarrow g_n Fetus

⑤ ASSE of acute Viral Infection in fetus

↳ Amniotic Fluid Polymerase chain Reaction. qq

In Acute Maternal Infection \Rightarrow 4 fold Rise in Ab titre in Paired sera.

— Avidity test.

⑥ Chorioamnionitis

| P.O.G. - | Amount of Amniotic Fluid |
|--------------|-----------------------------|
| 12 WK | 50mL |
| 16 WK | 250mL |
| 20 WK | 400mL |
| 32 WK | 1 Litre (Max ^m) |
| 36 WK | 900mL |
| Term / 40 WK | 800mL |
| 42 WK | 200mL |

POST-PARTUM HEMORRHAGE

(40)

3rd Stage \Rightarrow Placenta expulsion

\hookrightarrow Avg. time - 15-20 min

Prolonged $>$ 30 min.

Signs \Rightarrow ① Gush of blood;

Best sign

\leftarrow ② Lengthening of cord; \Rightarrow Apparent

③ Subpubic bulge; Permanent

④ Fundal height rise

Best \downarrow Placenta lying in vagina $>$ Lengthening of cord sign

⑤ Avg. Blood \Rightarrow Vaginal delivery = 500ml

Loss

C.S.

= 1000ml

Twinn vaginal delivery = 1000ml

PPH

1°

2°

\subseteq In 24 hrs of deliv

$>$ 24 hr upto 12 weeks

M/c cause \Rightarrow Uterine Atony

Retained
Placental tissue

Placental Abnormality
which is R/F for PPH \Rightarrow

$O=O$
"Succentriata"

$O-O$
"Bilobata".

4 Ts — Tone (Uterine Atony)

Trauma

Tissue (Retained placental tissue)

Thrombosis (defect)

* R/F for Atonicity → i) Multifetal ♀

ii) Macrosomia

iii) Polyhydramnios

iv) Induction of Labour

v) Augmentation of Labour

vi) Precipitate Labour

(onset - expulsion \leq in 3 hours)

vii) Any kind of APH

viii) Multiparity

ix) Diabetes Mellitus

x) Pre-eclampsia

xi) Chorioamnionitis

* Most important things for placental separation

↳ Uterine contraction.

Plane of Placental separation →

~~tk Q~~
Spongiosum

(4)

Methods by which placenta separates ↴

↳ Layer of decidua *



Placental separation

↳ Starts @ centre

Starts @ Periphery

Schultz Method*

- External - after complete bleeding Separation

begins = onset of Separation

- Blood loss ↓ (Total)

Total blood loss ↑

- Side presents @ vulva = fetal side

Mother had side



Shiny side
↳ Schultz * ↙ Shiny
80% Cases (M/c separation)

Shiny

↳ Duncan's Method*

20% Cases

- Retroplacental clot or is formed

- Absence of Retroplacental clot.

- * M/c of PPH ⇒ Atony > Genital tract Trauma > Retained tissue > Inversion > Rupture uterus > Amniotic fluid embolism.

Q&A

Prevention of PPH (AMTSL)

↳ by CHTO

Components ⇒

↳ Active Management of Third stage of Labour.

⇒ Give Uterotonic Agent + Immediately after delivery
↓
↓
≤ 1 min.

a) Doc!

Oxytocin

10 IU

I/m bolus

Onset
≤ 1 min

duration
3 hr

IV - Infusion Immediate 1 hr.

↳ Not give bolus b/c it is

R/F for = Hypotension

↓

Reflux Tachy. | Arrhythmia | MI | cardiac Arrest

• OXYtocin causes
Release of PGF_{2α}
from decidua.

- Naturally synthesizes oxytocin ⇒ Nonapeptide

- Artificially synthesizes oxytocin ⇒ Octapeptide

- Synthesized from - hypothalamus

↓

Pitaventricular Nucleus

- $t_{1/2} = 30 \text{ min (3-5 min)}$

- Stored in cold chain (2-8°C)

↓

b) Methergin (Ergometrine)

↳ Oxytung if/m

(42)

don't give i/v → causes transient severe hypertension

so; C/I m → Pre-eclampsia

Eclampsia

CVS disease

Peripheral vascular disease

Tetanic contraction (Acts more on LUS; while oxytocin on all uterus).

Brown colour - b/c of photosensitive nature.



c) SynoMetrine (5 U Oxytocin + 0.5 mg Methergin)

- Very Potent
- Not Doc → expensive
- Not available



d) Oxytocin = Synthetic oxytocin

↳ Octapeptide / Longer t/l/e

(-)

↳ 100 µg slow i/v over 1 min.



e) Misoprostol = PG_{E1} analogue



Prophylaxis = 600 µg (per oral)

Route = oral (In India \Rightarrow per Rectal)

Asthma is Not a C/I.

M/c side effects \Rightarrow Hyperpyrexia (fever & chills).

↳ directly proportional to dose

Other side effects \Rightarrow Nausea; Vomiting; Abd. Pain;
Hypotension.

II) Delivery of placenta by controlled cord traction!

eg "Klaus" Modified Brandt-Andrew Method

Rt. hand \Rightarrow Hold cord \downarrow do only when trained birth staff present.
L. hand \Rightarrow Push Fundus up.

III) Delayed cord clamping \Rightarrow



≥ 60 Sec. (1-3 min)

Gives More blood to fetus (80ml)

\uparrow Hb by 2 gmy. \leftarrow 50 mg Fe

Never done due to
AMTSL in Normal Labor.
early cord clamping

\downarrow In 60 sec.

Indications \Rightarrow

Baby Needs
Resuscitation

Rh Incompatibility

Baby is known case
of Heart disease

In HIV patient \Rightarrow Delay cord clamping done

Transmission happen during Labour.

(43)

IV) Intermittent Uterine tone assessment \Rightarrow

Uterine Massage = Not done

- Q Most imp. component ?? \hookrightarrow Not a component of AMTSL.
 (A) I; (B) II; (C) III; (D) IV

Management of PPH : (Shock Index = $\frac{\text{Heart Rate}}{\text{SBP}}$)

Symptomatic T/t :

- I) 2 Large bore ilv cannula
(14/16 Gauge)

- II) ilv fluids - crystalloids

- III) Arrange blood & blood products;

- IV) Catheterize - Urine output

- V) Blood group / Rh / CBC / Coagulation profile

\downarrow
to decrease Morbidity & Mortality Rate.

BiManual

T
B/M

Uterine Massage

+
call for help

+
Uterotonics given

\hookrightarrow doc Pit

\hookrightarrow oxytocin

\downarrow
20IU \rightarrow 40IU / 500ml of

NS / RL

Not in 5% dext.

\downarrow
If we give Large doses

\downarrow
Large duration

\downarrow
Electrolyte deficient media

\downarrow

\downarrow
Result in water intoxication.

↓ if Not Responding

Inj. Tranexxa (Tranexamic Acid)

↓

↳ Anti fibrinolytic drugs

Anti fibrinoly

1gm slow

↓

↓ if Not Responding

Methergin
0.2 mg (i/m)

carboprost (Methyl Prostaglandin)

↓

0.25 mg (i/m)

↳ H/o Asthma → Not given; Pt. dies off
bronchospasm

↓

Diamox (M/c side effect)

↓ if Not Responding. of carboprost)

Misoprostol → to be used i/m; i/v can't be given.

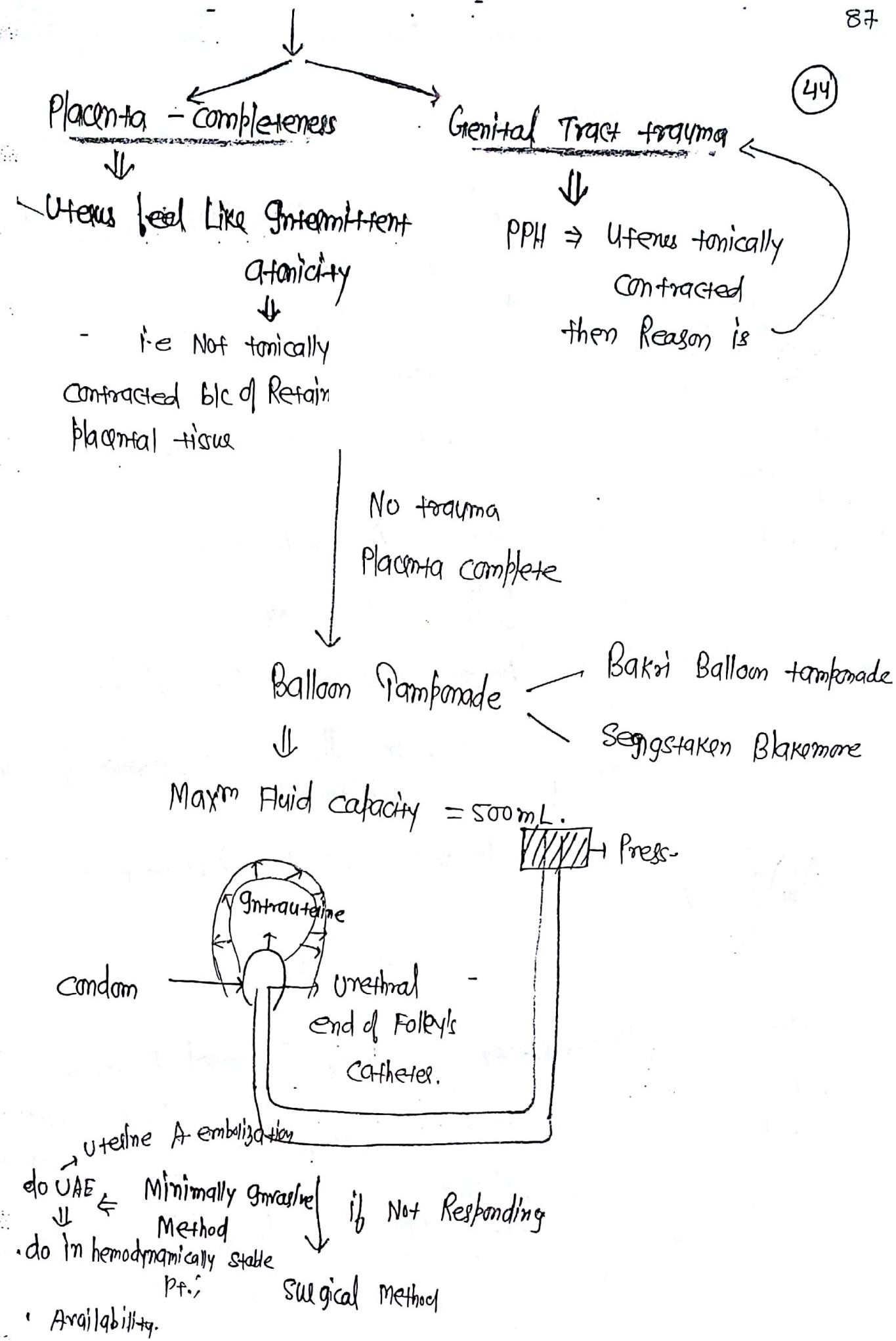
↳ Tit = 800mcg (Sublingual)

Not given P/V (Per vagina)

Can give P/R (Per Rectal)

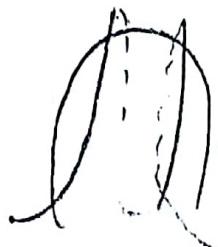
* All drugs trial to happen after 30min.

↓ if Not Responding.



Surgical Management

- Uterine compression suture / β -Lynch suture $\xrightarrow{\text{Brace}}$



Applied on uterus & helpful
only in Atonic PPH

↓ if Not Responding

- B/L Uterine A Ligation

↓ if Not Responding

- B/L - Anterior division of Internal iliac

{
↳ 5 cm distal to bifurcation of common iliac

+ Yes Pelvic Pulse Pressure by syst.

↓ if Not Responding

- Hysterectomy (Possible to do Sub-total hysterectomy)

{ Remove uterus - saves the
Kloss "Subaracnoid" cervix

{ If bleeding
continues thinks
about "D.I.C."

↳ to Improve sexual life of female

↳ DIC **

TRAUMA → Perineal tear differs from episiotomy (45)

to prevent

① do Routine episiotomy ⇒ No

-② one hand = Support the perineum

③ other hand = Maintain flexion of head

④ tell the Mother Not to push @ the time of head delivery.

⑤ NICE → application of warm Guidelines Perineal compresses

It is surgically planned Incision.

done in special cases

Forcesps; Breech cond'n

MedioLateral

Median

↓

Extends

↓

Rectum sphincter & Mucosa

↑ Pain

↑ Dyspareunia

breaks down easily

↑ Blood loss

Poor cosmetics

degree of Perineal tear ⇒

Repaired 1st degree — Vaginal Mucosa & Skin
In Labour

Room. 2nd degree — Perineal Muscle

3rd degree — A — Less than 50% EAS torn

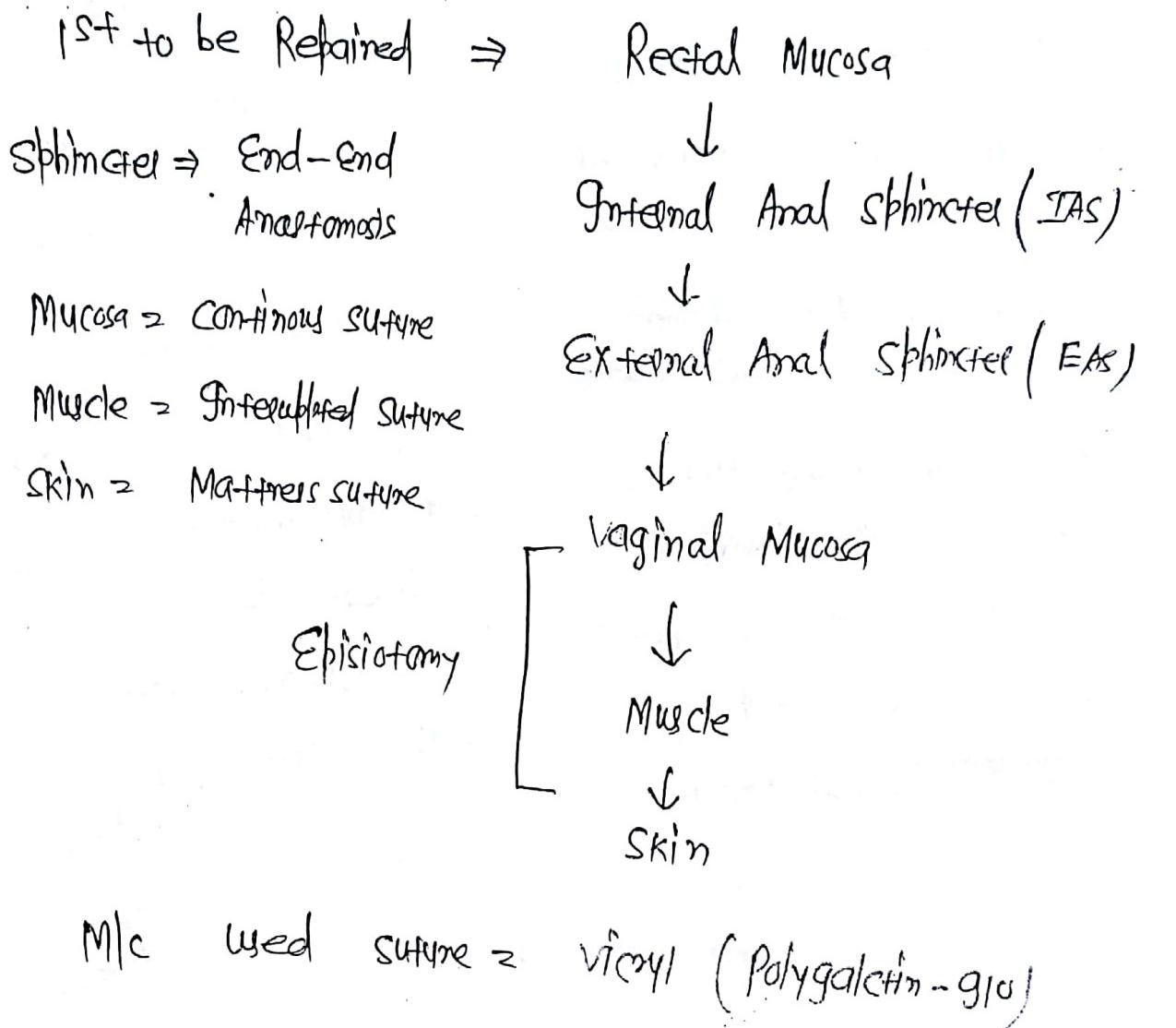
B — More than 50% EAS torn

C — Both IAS & EAS torn

complete
Perineal tears

4th degree — Up to Rectal Mucosa

Complete Perineal tears \Rightarrow obstetric emergencies
in 24 hrs
 \hookrightarrow 3 weekly



* Another Tear \Rightarrow Hematoma

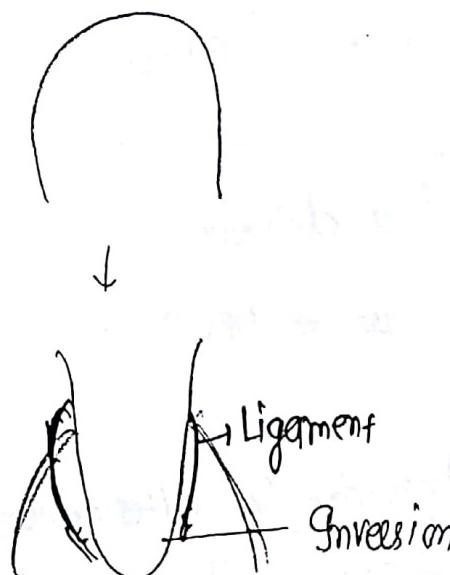
\hookrightarrow M/c Presentation = Pain
• Inability to pass urine

$\uparrow H_2$ Analgesics
Monitor
ace-bancks (Local anesthetics) \rightarrow vasoconstriction

```
graph LR; AnotherTear[Another Tear] --> Hematoma[Hematoma]; Hematoma --> MCPresentation["M/c Presentation = Pain"]; MCPresentation --> Inability["Inability to pass urine"]; Inability --> Analgesics[Analgesics]; Analgesics --> Monitor[Monitor]; Monitor --> AceBancks["ace-bancks (Local anesthetics)"]; AceBancks --> Vasoconstriction[Vasoconstriction];
```

- Looks like bluish tender swelling
- Surgical Mx =
 - ① Shock
 - ② expanding In size
 - ③ excruciating Pain (hematoma expanding internally)
↳ Muscle.

* In Inversion \Rightarrow Stretch on the Ligament



\downarrow
Neurogenic shock (1st shock to develop)

\downarrow
Hemorrhagic shock

\downarrow
death.

Rx \Rightarrow do Manual Repositioning
(Johnson's Method)

Part which comes out Last;
has to be Reposited first.

\downarrow
ib balls

Hydrostatic Method (O'Sullivan Method)

\downarrow
ib balls

Surgical management

Surgical Management for Inversion

Haultain
Huntington
Spinelli

- Emergency; give general Anesthesia; Not spinal



Anesthesia

Relaxes Uterus

also in Inversion

Manual Removal of Placenta

Hemodynamically Unstable patient

Q&A Pt. goes into shock ^{of death} after delivery

Most probable cause \Rightarrow PPH

Q&A Pt. goes into shock immediately after delivery

- Most probable cause \Rightarrow Inversion

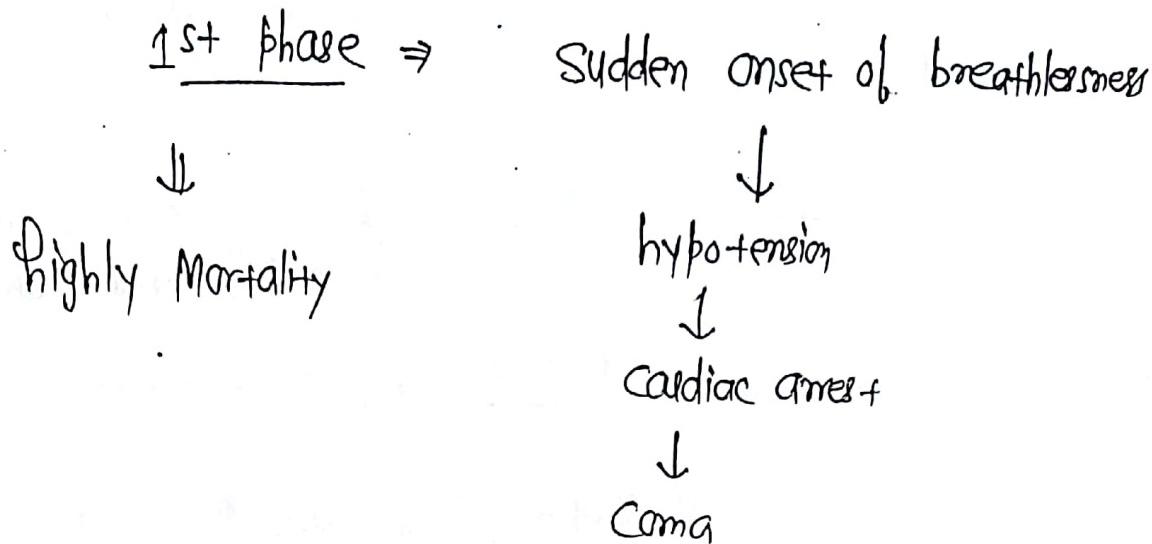
Q&A Pt. goes into shock (unexplained) after delivery

Most probable cause \Rightarrow Amniotic fluid embolism

(Very Rare / diagnosis of exclusion)

K/o "Anaphylactoid syndrome
of Pregnancy"

No Lab test to confirm or refute diagnosis. 93
* Clinical diagnosis of AFE ↴ (47)



2nd Phase ⇒ Pt. goes into DIC + Hemorrhage
↓
death seen

If we can do ⇒ Sample from Pulmonary vessels

↓
Lamugo / Vernix / amniotic fluid seen

DIC

MCC = - Abruption

also by = AFE

Massive Hemorrhage (APH / pph)

Sepsis (Septic abortion)

GVD (≥ 4 weeks vs Risk of DIC)

Management of GUD \Rightarrow wait & watch
 \Downarrow
Must go into spont. Labours \subset in 2 weeks

\rightarrow signs \Rightarrow Robert's sign \Rightarrow comes @ 12 days
↳ gas in Major blood vessels

Spalding sign \Rightarrow comes @ 7 days

Ball sign \Rightarrow Hyperflexion of spine

Buddha \Rightarrow Subcutaneous edema

sign

↳ seen in hydrops fetalis

\rightarrow Not a sign of GUD.

PRE ECLAMPSIA

48

- * Gestational HTN \Rightarrow
 - i) BP $\geq 140/90$ on 2 occasions; 4-6 hr apart
 - ii) BP high > 20 weeks;
 - iii) Return to N \leq in 12 weeks Post-partum.
 - iv) No evidence of Proteinuria
 - * Blood Pressure Should taken in sitting position.
 - * 5th Korotkoff sound heard
 - * Pre-eclampsia \Rightarrow
 - i) BP $\geq 140/90$ on 2 occasions; 4-6 hr apart
 - ii) BP > 20 weeks
high
 - \exists Any of the following

Proteinuria

$= \geq 0.3 \text{ gm/300 mg}$

In a 24 hr Urine Sample

or

Urinary $\frac{\text{protein}}{\text{creatinine}} > 0.3$

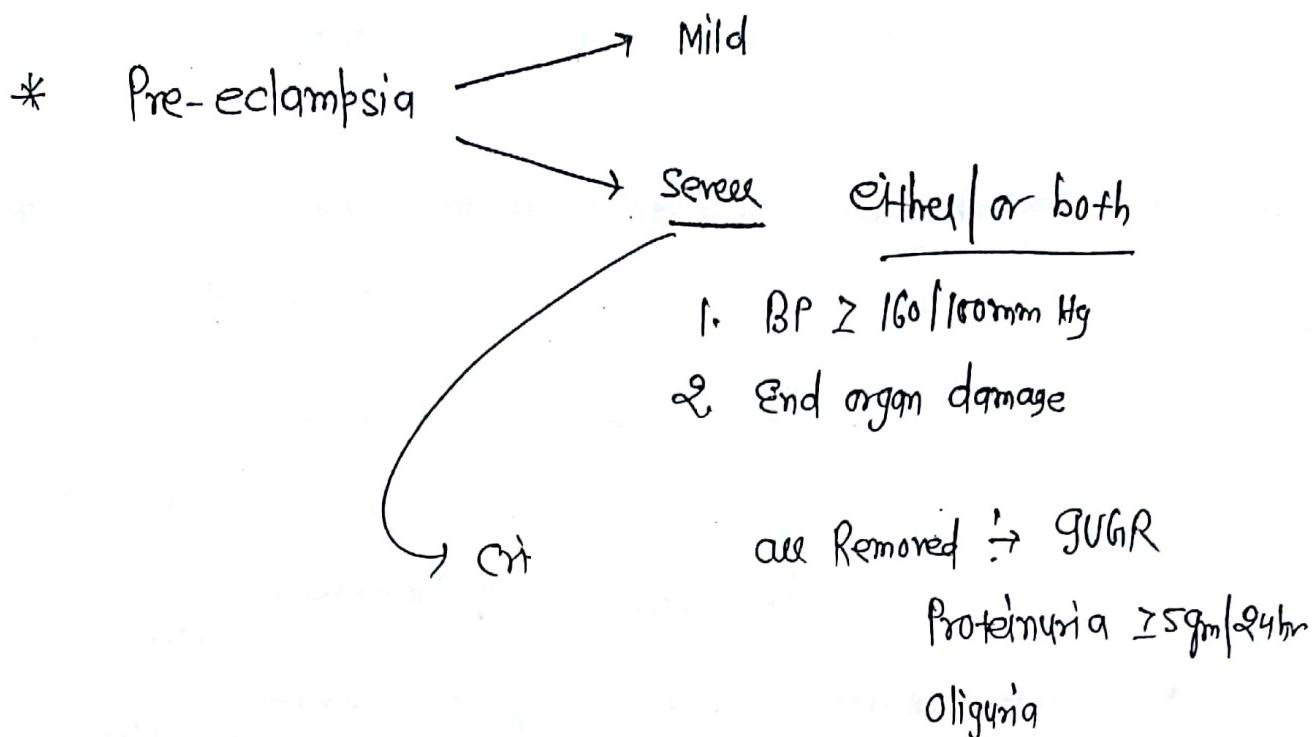
OR

Dipstick (+)

End-organ damage

(It could be Any of the following)

 - i) Platelet count $< 1 \text{ Lakh};$
 - ii) Serum creatinine > 1.1
 - iii) Liver enzymes all More than twice the N value
 - iv) Pulmonary edema
 - v) Cerebral / visual symptoms



* Eclampsia \Rightarrow Pre-eclampsia + Seizures

* Chronic HTN in ♀ \Rightarrow i) BP is high before conception;
 ii) BP is high in 1st 20 wk;
 iii) BP Remaining high $>$ 6 weeks post partum period.

* Chronic HTN & superimposed Pre-eclampsia \Rightarrow
 i) New onset proteinuria beyond 20 weeks
 ii) End organ damage $>$ 20 weeks
 iii) Uncontrolled HTN $>$ 20 weeks

- Gmbanding eclampsia \Rightarrow if she has Any of the following sign & symptoms \Rightarrow i) epigastric pain (49)

↳ stretching of Liver

Capsule.

Spontaneous subcapsular hematoma

ii) Headache/ dizziness

↳ cerebral hypoxia

iii)

Blurring/ diplopia/ Blindness $\xrightarrow{\text{Blindness}}$ central occipital lobe hypoxia

Scotoma

$\xrightarrow{\text{Peripheral}} \text{Retinal detachment}$

Hypertensive Retinopathy

iv) HELLP Syndrome

↳ Mostly - Feature of severe Pre-eclampsia

↳ Blood pressure in 15% of patient is (N)

Criteria

$\Rightarrow \left\{ \begin{array}{l} H = \text{Hemolysis. (a) Peripheral Smear} \\ \quad \downarrow \text{Schistocytes} \\ \quad \downarrow \text{(Helmet cells);} \end{array} \right.$

b) S. Bilirubin ≥ 12

E \Rightarrow Elevated Liver enzyme ($ALT \geq 70 \text{ IU}$)

P \Rightarrow Low Platelet count ($\text{Plt. count} < 10,000$)

all should be in patient

* M/c Presentation \Rightarrow Pain \rightarrow epigastric pain
(Rt. upper quadrant)



seen in 3rd trimester

~~①~~ \Rightarrow ~~i) Acute Fatty Liver of Q (closest to HELLP)~~
of HELLP Sx ii) Hepatitis

iii) obstetric cholestasis

differentiated by presence of

- ① Hypoglycemia
- ② Hepato Renal Sx
- ③ Coagulation defect
- ④ Pancreatitis

Pathophysiology \Rightarrow i) (AbN) of β -oxidation \rightarrow Mother (Mitochondrial)
Mainly in 3rd trimester of Fatty acids

(More severe form
Liver Gravy)
by Postmortem Liver Biopsy confirmed.
ii) LCHAD enzyme deficiency \rightarrow Fetus

M/c cause of Acute Liver failure in Q = AFLP

- High Mortality Rate

* Acute hepatitis

↳ have Prodromal symptom.

(50)

- Liver enzyme Raised

- Bilirubin - Markedly Raised

M/c Acute hepatitis in ♀ ⇒ Hepatitis E

↳ high Mortality Rate

* Obstetric cholestasis

- M/c Symptom ⇒ Pruritis

- Seen in 3rd trimester

- Estrogen

- Mutation in Genes ABCB4

- ABCB11

- Serum bile acids become accumulating

- ↳ Diagnostic test

- RH ⇒ Ursodeoxycholic Acid

Most Risky for fetus = Pre-term Labour;

Constitute the bile acids; ⇌ Sudden still birth;

Result in the all of
three.

Mecominum Afiltration syndrome

Termination of ♀ ⇒ (37 weeks) → (38 weeks)
 In obstetric cholestasis

* * Recurrence Risk of HELLP = 4-7%.

Obstetric cholestasis = 70%.

* Pathophysiology of Pre-eclampsia ↗

Vascular Remodelling

Absent

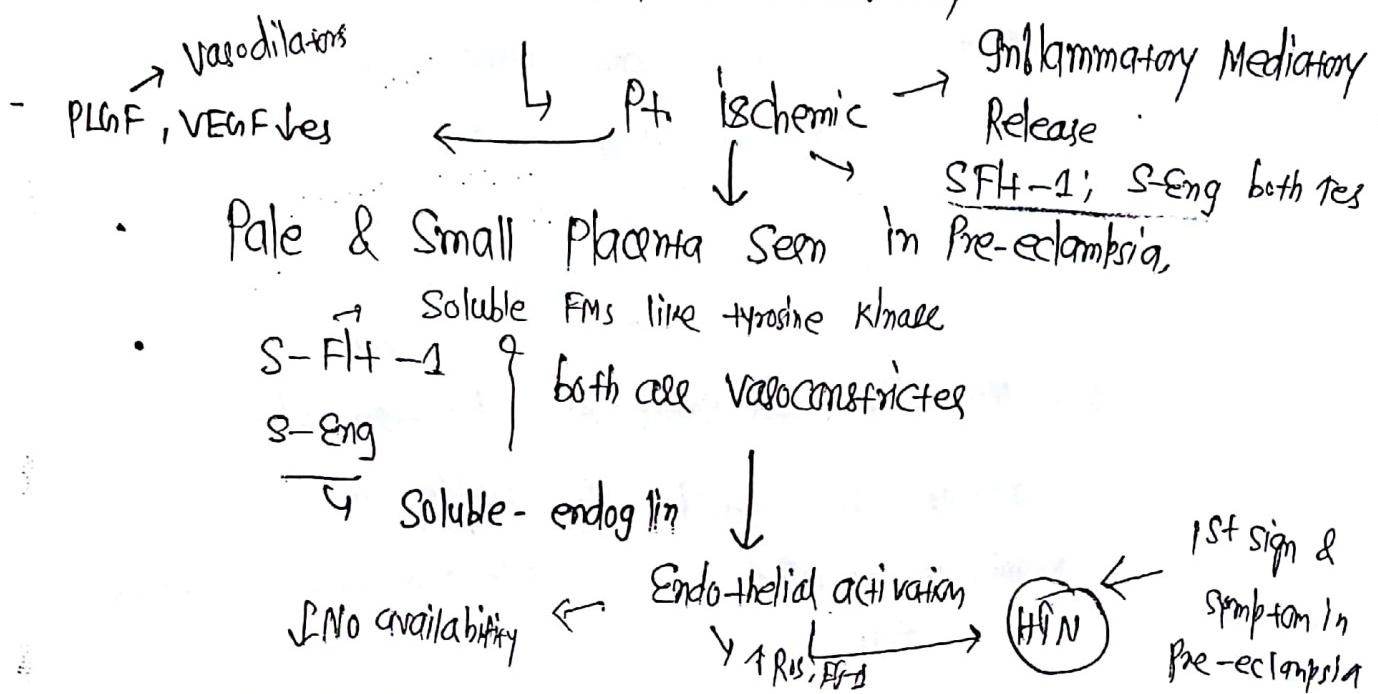


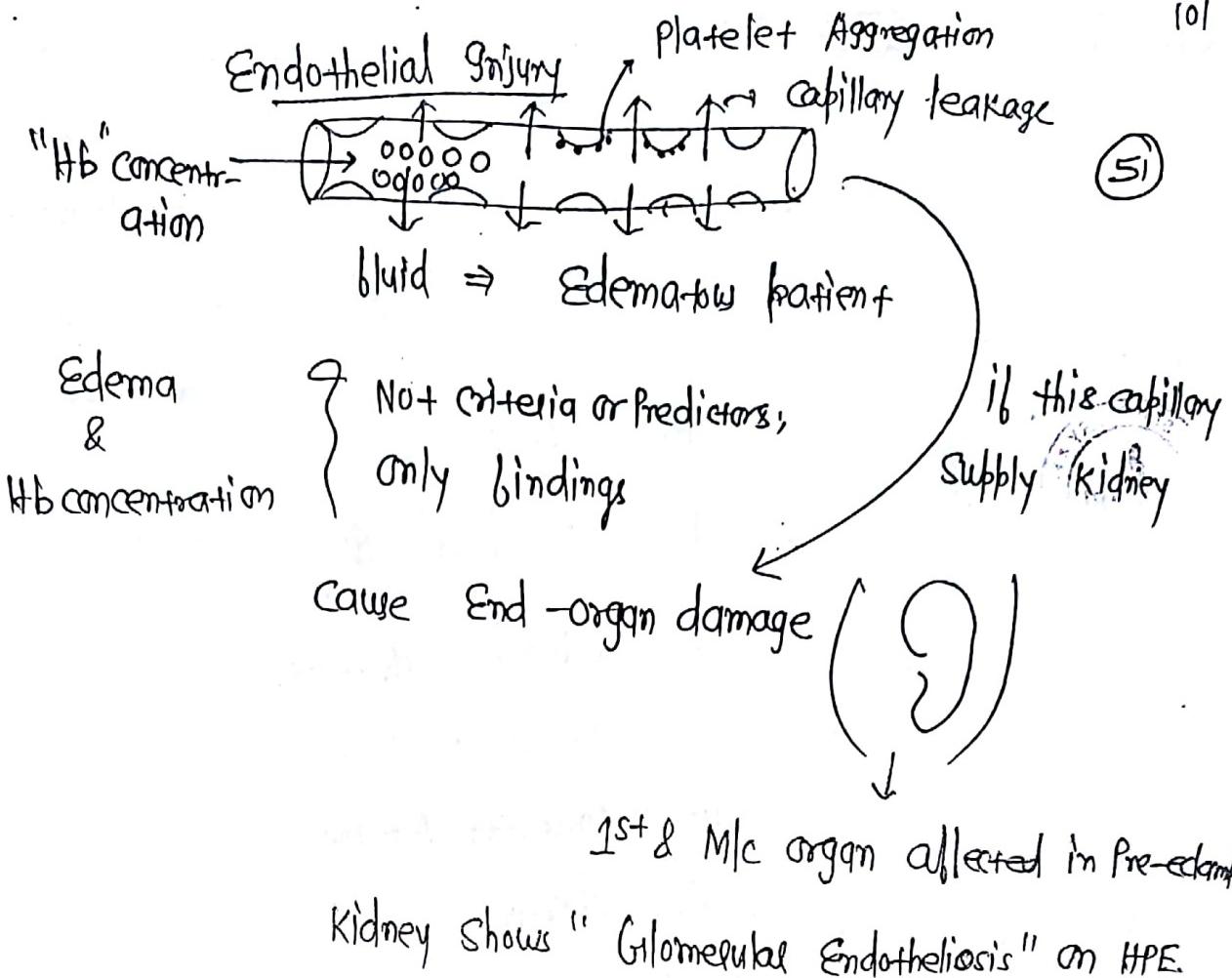
Vessels in Decidua Basalis



↳ it means remain sensitive to vasoressin

Result in Utero-placental Insufficiency





↑ Serum Uric acid \Rightarrow also a ~~binding~~ ^{binding} of criteria

* Immune theory \rightarrow

- Normally $\Rightarrow \downarrow T_{H_1} \longrightarrow \uparrow T_{H_2}$ Response

In pre-eclampsia - this shift doesn't happen,

\Downarrow

so: $\uparrow T_{H_1}$

\Downarrow

Altered Immune Response to paternally derived Antigen.

- Risk factor \rightarrow
- i) Primigravida; (b/c 1st time exposed)
 - ii) Molar pregnancy (Extra paternal chromosome)
↳ can develop Early onset Pre-eclampsia!
 - iii) APLA
 - iv) Multifetal ♀
 - v) Chronic Hypertension
 - vi) Renal disease;
 - vii) Diabetes Mellitus;
 - viii) Obesity
 - ix) extremes of age $< 18, > 35$ years
 - x) Previous H/o Pre-eclampsia

- Protective factor \rightarrow
- i) Smoking
 - ii) It has +ve association \in Placenta previa;
It has +ve association \in Abruptio.

- Predictors of Pre-eclampsia \rightarrow
- i) \uparrow SFlf-1
 - ii) \uparrow s-eng
 - iii) \downarrow PLGF
 - iv) \downarrow VEGF
 - v) Increase in Doppler
 - vi) \downarrow Urinary cathecolamine excretion,

* Uteline A. Goffles

(52)

(N) → Notching - disappear by 22 weeks

Persistent Notching — beyond 22 wk — Predictor for Pre-eclampsia

Prevention of Pre-eclampsia → Aspirin

↓
150 mg — once in a day
start in 16 weeks
↳ continue throughout Q
only indicated for high Risk patient for Pre-eclampsia

Ca Supplementation

↳ Prevent Pre-eclampsia only in women who have cat2 deficient

- Salt Restriction
 - Omega 3 Fatty acids
 - Vit. C, E
- } No Role in prevention.

* Management of Pre-eclampsia \Rightarrow

Symptomatic

- Anti-hypertensive
- Anti-seizure

Specific

- Termination of ♀

Stop progression

Reverse the damage

Anti-hypertensive

i) When do you start Anti-hypertensive

↳ If BP Persistently $\geq 150/100 \text{ mm of Hg}$

ii) Which drug - Doc

↳ Labetalol ($\alpha + \beta$ -Blocker)

but; In Chronic Hypertension

↳ Methyl dopa (safest Antihypertensive in Pre-eclampsia)

Doc for Acute HTN in ♀ ($\geq 160/110$)

↳ acc. to ACOG; Any of these we can 1st line drug

① \downarrow
i/v Labetalol

>

② \downarrow
i/v hydralazine

>

③ Sustained Release Nitroprusside

IV Labetalol



20 mg IV

↓ after 10 min

40 mg $\xrightarrow{\text{after 10 min}}$ 80 mg \longrightarrow 80 mg upto total 300mg/24hr.

(53)

(4) i/v Nitroglycerine \Rightarrow used in Pulmonary edema

(5) If Refractory Hypertension \Rightarrow Sodium Nitroprusside
Anti-hypertensive drugs cause (cyanide toxicity)

* Methyl dopa is Not given in Acute HTN
↳ b/c

iii) drugs not to be used as Antihypertensive \rightarrow

i) ACEi;

ii) ARBs;

iii) Diuretics;

iv) β Blockers;

v) Digoxide

IV Target Blood Pressure \Rightarrow

Systolic blood pressure = 120 - 130 mm of Hg

Diastolic blood pressure = 80 - 90 mm of Hg

TERMINATION OF PREGNANCY

Gestational Hypertension / Mild Pre-eclampsia ; well controlled BP
↳ @ 37 weeks

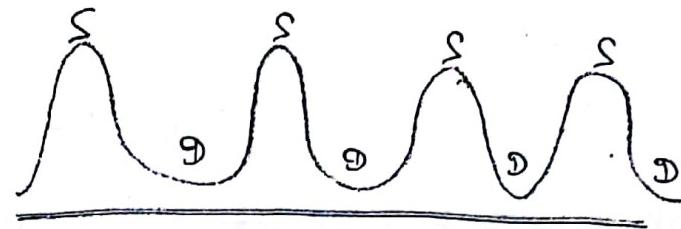
Severe Pre-eclampsia — well controlled - NoP - @ 34 weeks

* Indication of Termination of ♀ Irrespective of Gestational age,

- i) Impending Eclampsia;
- ii) Eclampsia
- iii) HELLP Syndrome;
- iv) Abruption / Fetal distress
- v) Uncontrolled HTN / Rising serum creatinine
- vi) RDSF - Reversal of End diastolic flow
↳ Doppler of Umbilical Artery.

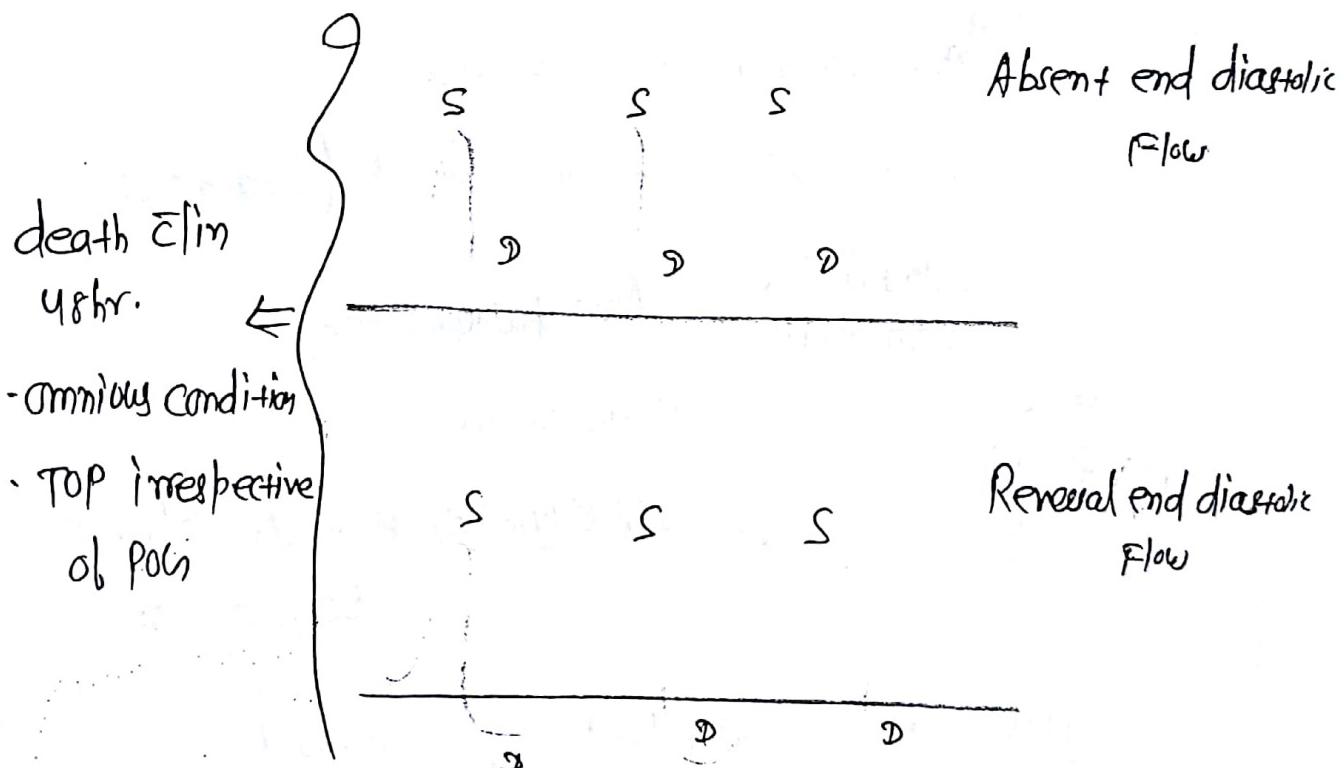
(N) Umbilical Artery Flow $\Rightarrow \frac{S}{D}$ Ratio

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In (N) Pregnancy $\Rightarrow \frac{S}{D}$ Ratio less as Pulses

In UPI $\Rightarrow \frac{S}{D}$ Ratio less ($\geq 30 \frac{S}{D} \geq 3$)



* Whenever Possible do Vaginal delivery > Cervical section
(High Risk)

Anesthetic complication
blood loss

* Gn G.S.

↳ epidural Anesthesia given

↳ if Not given then

Mark Neuraxial Anesthesia

Never give General Anesthesia

ECLAMPSIA

Preeclampsia + Seizures

↳ GTCS (Grand mal)

1st Seizure ⇒ Antepartum (M/c)

Intra partum

Post partum ⇒ If Means clin 48 hrs
Seizure comes

Causes ⇒ Cerebral hypoxia + Cerebral edema

Rx ⇒ MgSO₄ ⇒ Central Action

doesn't do any peripheral vasodilation NMDA Receptor ↙ ↘ Cerebral vasodilation

* In Periphery $MgSO_4 \rightarrow C_g$ channels

(55)

So; don't club Ca^{2+} channel blockers (for HIN) & $MgSO_4$

↓

Causes "Neuromuscular Paralysis"

So; $MgSO_4$ gives & Labetalol ($\alpha_{1,2}^{-1}$)

*

1stly - looks for vitals

↓

Stabilize the position (by tie her legs & hands)

↓

Secure Airway — Mouth GAG

Suction

Oxygen by Mask

Gm. $MgSO_4$ + Anti-HIN

Pritchard's Regimen

Loading dose

14gm

↓
4gm - slow i/v @ 1gm/min
10gm - i/m or csm in each buttocks

Monitor by Heart Rate

Maintenance dose given every 4 hourly until ≈ 48 hr
↓
before giving dose check \Rightarrow Patellar Reflex (+) if $RR > 12$ min

↓
then give 5gm i/m dose
(@ alternate site) \Rightarrow Urine output $> 30ml/hr$ to check absorption

16 Any of the

| | | |
|--|---|----------------------------------|
| Patellar Reflex \oplus $RR > 12/\text{min}$ $\text{Urine output} > 30\text{ml/hr}$ | } | any Absent ↓ ↓ if the dose |
|--|---|----------------------------------|

Send serum Mg^{+2}

↓
Therapeutic Level = 4-7 meq

| | | | |
|----------------------------|---|--|---|
| \textcircled{a} 9-12 meq | } | @ 10 meq = Patellar Reflex Absent @ 12 meq = Respiratory distress | ↓ warmth, diaphoresis; slurring of speech |
|----------------------------|---|--|---|

Antidote for $MgSO_4 \Rightarrow 10\text{ml of } 10\% \text{ Ca glucomate}$

* Indication of Prophylactic $MgSO_4 \Rightarrow$ In Impending Eclampsia,

In HELLP syndrome;

In Severe Pre-eclampsia

* Oliguria is Not a toxicity symptom of $MgSO_4$.

Q&Q Primigravida 34 weeks gestation; ~~Essentiality~~ = headache X 4 hrs; 2 episodes of vomiting O/E BP=160/110 mmHg; FHR-N; Most appropriate step of Mx (S6) Fundal height = 34 weeks; Urine output protein (+).
 (Firstly) MgSO₄ + Anti-HFN + Top
 () → then → then

* Eclampsia is indication for TOP
 ↓
 vaginal delivery > C.S.
 delivered \leq 1m 24 hours

DIABETES IN PREGNANCY

MELLITUS

Overt

Pregestational

Known case of "diabetes
Mellitus"

Gestational

1st time deranged
Sugar all↑ in pregnancy
diagnosed

- Most common

Single Step

↳ both screening & diagnostic

2 hr OGTT

- National guideline DIPS^I criteria

- done @ 24-28 weeks

- glucose load 75 gm (oral)

- ± Fasting

after 2 hrs

75 gm oral
glucose

Blood sample

> 140 - GDM

> 200 - overt GDM

120-140 = Impaired glucose tolerance
of pregnancy

* All ♂ Women Must undergo & have OGTT

↳ Universal Screening

(57)

- * Early testing — 1st Antenatal visit
- ① if she gives H/o Baby \in GCA
- ② if she gives H/o Still birth
- ③ if she gives H/o Macrosomic baby
- ④ if she gives H/o Diabetes — 1st degree
- ⑤ if she is obese

180 f

FBS
or

HbA_{1c}

Fasting $\geq 126 \Rightarrow$ overt diabetes.

* Diabetes \Rightarrow High Risk

For Foetus

↳ ↑ Risk of Gross Congenital Anomaly by four fold In compare to ♂ ♀

↓
di^t hyperglycemia (leto-toxic)

↓
Free Radical Injury

- * Risk of GCA rises in oneself b/c gestational tes after 24-28 weeks
 - * M/c GCA \Rightarrow CVS > NTD
 - * Most specific \Rightarrow Caudal Regression Syndrome (Sacral Agenesis)
 - * M/c CVS Anomaly in fetus of diabetic Mother \Rightarrow VSG
 - * M/c Specific Anomaly in fetus of diabetic Mother \Rightarrow TGA
 - * M/c finding in fetus of diabetic Mother \Rightarrow HOCM

ANSWER: Anencephaly

Frog eye design | Mickey Mouse sign on us,

Anencephaly — earliest \Rightarrow 10 weeks

→ diagnosed \Rightarrow 14 weeks

- * Max^m complication seen in female foetus except →

(58)

Macrosomia } In Male
Respiratory complication }

- * Anencephaly seen in Polyhydramnios ♀

↴ Post-term Labour > Pre-term Labour
 (Initiation of Parturition ⇒ CPM)
 ↴ Mainly Face Maldevelopment seen.

- * Bernama sign } Seen in spinal
Lemon sign bifida & Arnold-Chiari syndrome.

- Test - for Risk of Anomalies

HbA_{1c} ⇒ @ 6.5 ⇒ Risk res by 3%.

@ 7.5 ⇒ Risk res by 4%

@ 8.5 ⇒ Risk res by 8%

> 9 ⇒ Risk res by 15%

- * USh ⇒ for Anomalies diagnosis → Palpat[→] Imaging for fetal anomalies
↳ 18-20 weeks ↳ Level-II (TIFFI)

* Level-II USG do for all preg. women

Prevention of Anomaly ↴

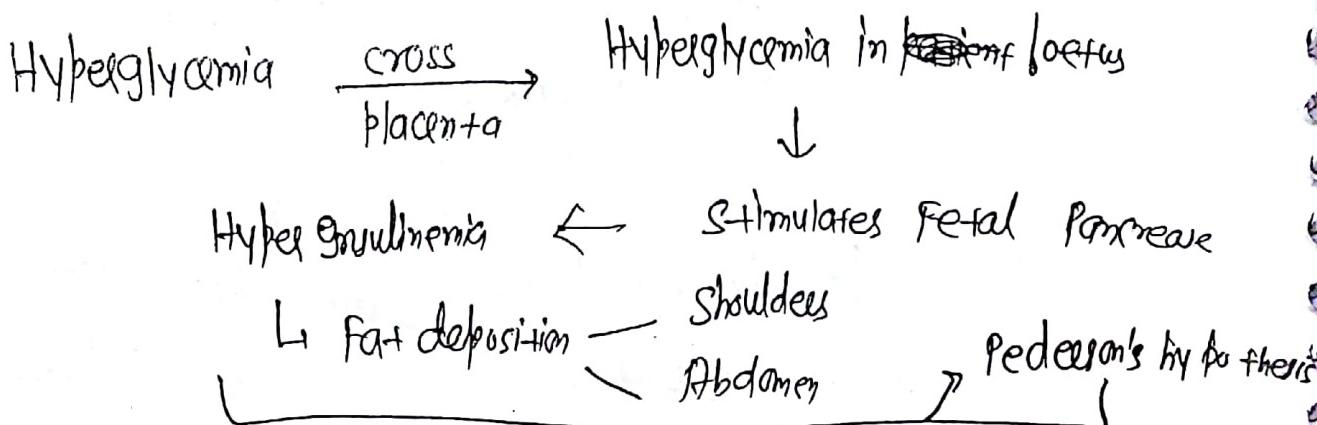
if we know that pt. is overt diabetic → Pregnancy - give Insulin
tight glycose control

give Folic Acid Supplementation Prophylaxis of SFA
overt diabetic ↓ 4mg ^{NID} 0.4mg 0.5mg
previous H/o NID ↓
Antiepileptics ↓

When to start! → as soon as they
Folic acid present preconceptionally

* Minⁿ Requirement: 1 month before to 3 month after

2. Macrosomia



Diagnostic for Macrosomia

(59)

EBW \geq 4Kg



On USG

L Single - Fetal growth



Abdominal circumference (Macrosomia)
IUGR

* USG also uses to calculate Gestational age

Best In 1st Trimester for Gestational age = CROWN-RUMP Length

2nd " = Biparietal diameter

3rd " = Femur Length

Best for Gestational Age = CRL

* Best for Gestational Age \rightarrow

(A) CRL for 16 weeks

(B) BPD @ 16 weeks

(C) Femur Length 30 weeks

(D) Abdominal circumference 30 weeks

* Macrosomia Patient presents to Shoulder dystocia

↳ 1 min delay in the delivery of shoulders after delivery of head



TURTLE SIGN



↳ sudden pulling back of head towards Maternal Perineum

* Mx of Shoulder dystocia ⇒

H - call for Help

E - empty bladder [episiotomy do]

L - 1st - Legs - Mc Robert's Manoeuvre

P - Suba pubic pressure ↳ sudden flexion & Abduction of Maternal thigh on the Maternal Abdomen

Not fundus pressure

↳ ↑ space in A-P diameters

E - Enter - Woods screw Maneuvre

OR ↓

R - Removal of Posterior Arm

↳ We injured Lateral Cutaneous Nerve of thigh

R - Roll over on 4 limbs

↓

Gaskin all fours

↳ Metalgia Paresthesia

Last Maneuver ⇒

Zavanelli Maneuvre

↳ Push head back & do C.S. into mother

- * Theoretically - destructive procedure -
 - Symphysiotomy
 - cleidotomy 60
 - Gastric # clavicle

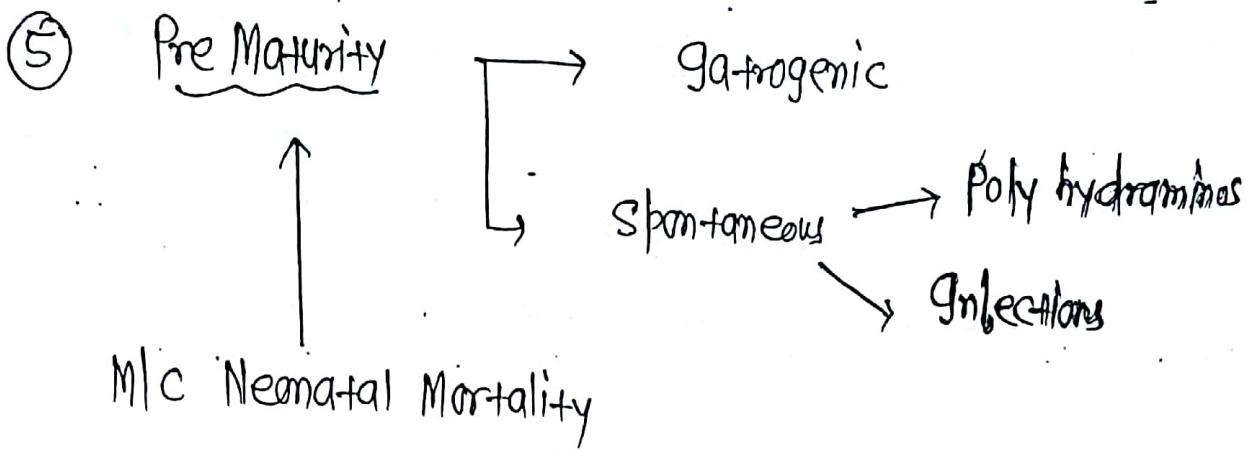
- * M/c fetal injury in shoulder dystocia \Rightarrow Brachial plexus
- * M/c maternal complication in shoulder dystocia \Rightarrow PPHT

3) GUGR \Rightarrow Rare (common)

↴ In diabetic vasculopathy } Utero placental insufficiency
 Pre-eclampsia

↴ Expected birth weight < 10th percentile

④ Still birth \Rightarrow Seen in Macrosomic baby, Male baby
 (↑ O₂ requirement)
 ↓
 Hypoxic episode
 ↴ Sustained — still birth



⑥ Lung Maturation is delayed

- ↳ Phosphatidyl glycerol
- Not L/S Ratio

⑦ Operative delivery

- ↳ ↑ Risk of Respiratory Problem

⑧ Immediately after delivery

- i) \rightarrow Hypoglycemia
- ii) \rightarrow Polycythemia
- iii) \rightarrow Hyperbilirubinemia
- iv) \rightarrow Hypocalcemia
- v) \rightarrow Hypermagnesemia } Prematurity
- vi) \rightarrow RDS
- vii) \rightarrow HOCM

* Maternal complication of Diabetic Mother \rightarrow

i) Abortion \Rightarrow Uncontrolled diabetes (61)

ii) Polyhydramnios $\xrightarrow{\text{causes}}$ GCA
 \rightarrow ↑ glucose in Amniotic fluid
 \rightarrow Hyperglycemia in fetus
 (Polyuria)

iii) oligohydramnios (uncommon)

seen in \leftarrow Diabetic vasculopathy & in utero-placental
 Pre-eclampsia Insufficiency

iv) Pre-eclampsia \Rightarrow ISF Risk Test

v) Infections $\xrightarrow{\quad}$ UTI

\backslash candidiasis

vi) Complication of diabetes $\xrightarrow{\quad}$ Vascopathy

$\xrightarrow{\quad}$ Retinopathy

$\left\{ \begin{array}{l} \xrightarrow{\quad} \\ \xrightarrow{\quad} \end{array} \right.$ Nephropathy

Pre-existing \Rightarrow worsen in pregnancy

viii Operative deliveries

Vaginal C.S.

viii) PPH (over distension)

ix) Pt. can develop tafel overt diabetes (in 50% cases)

follow up ↴ 6 week (Post partum visit) | 12 wk

by OGTT test

* Management of Diabetes Mellitus in ♀

Maternal Monitoring

- Blood Sugar Level

~~overdiabetes~~

↓

Pregnancy

1

Insulin

only diabetes die

Huf. = carbohydrate

$$45x = 1048$$

20% = $\frac{2}{10}$ or $\frac{1}{5}$

do Sugar profile ↓ after 72 hr

Fetal Monitoring

- Staff to Monitors - 3265k

DFMC (daily fetal Movement count);

NST

BPP (Biophysical Probe)

U.S.G.

Doppler Never use

only in

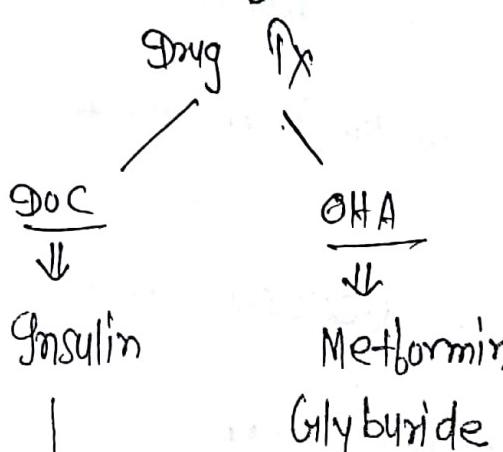
gut R UTI

Sugar Profile

Fasting ≥ 95 or

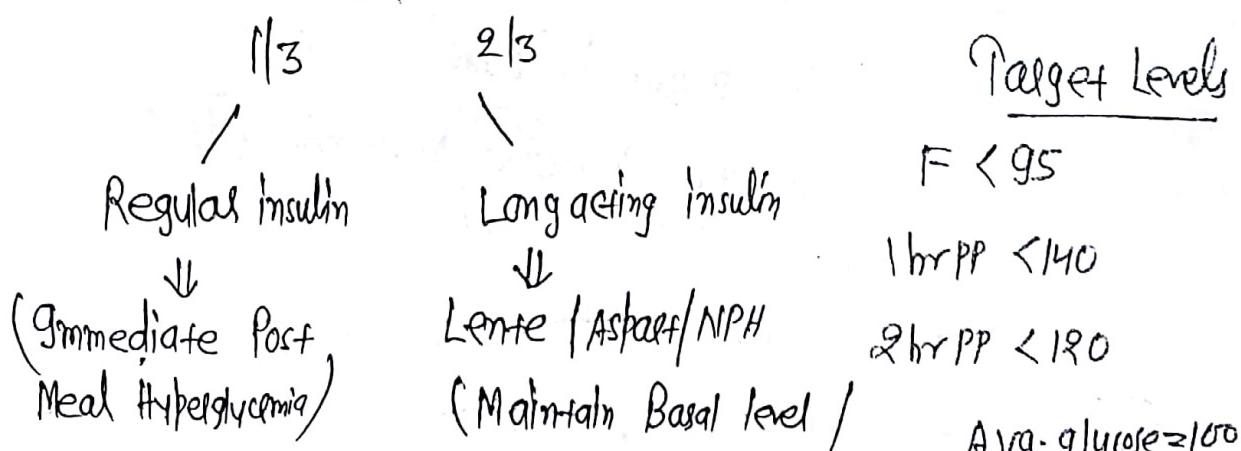
1 hr PP ≥ 140 or

2 hr PP ≥ 120



M/c Ginsulin use \Rightarrow Regular Ginsulin

Total dose



$$1-12 \text{ wk} = 0.7 \text{ U/kg}$$

$$12-28 \text{ wk} = 0.8 \text{ U/kg}$$

$$28-34 \text{ wk} = 0.9 \text{ U/kg}$$

$$\geq 35 \text{ wk} = 1 \text{ U/kg}$$

$$\text{HbA1c} \leq 6.5\%$$

* TOP = Gestational diabetes = EDD = 40 weeks
on diet alone

over+ | GDM on = 39 weeks
insulin

- * Diabetes (Alone) is Not an indication for C.S.
- * Expected birth weight $\geq 4.5 \text{ kg}$ in a diabetic pregnancy



Cesarean section

- * In Non diabetic pregnancy $\geq 5 \text{ kg}$

* Insulin Requirement in Labour \rightarrow Yes



Stop Insulin in Labour

& do Intensive Monitoring @ Qhrly.

ECTOPIC PREGNANCY

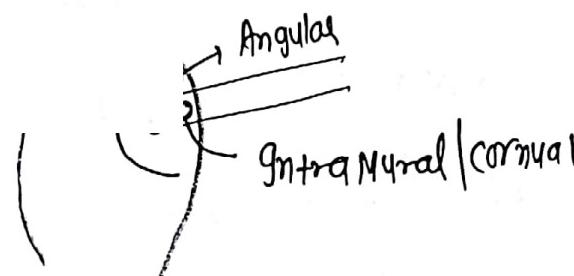
- Any Pregnancy is implanted outside the Uterine Cavity.

(63)

- Cornual Pregnancy → ectopic ♀

IntraMural part of Fallopian tube

- Angular Pregnancy → Intrauterine ♀
 ↳ glo of uterus



* Round Ligament is attached Lateral to the growth of Uterus

↳ Angular ♀

* Round Ligament is Medial to growth of uterus

↳ Cornual ♀

* Heterotopic ♀ ⇒ 2 Pregnancy simultaneously at different sites of implantation.

Most common ⇒ Intrauterine + Intratubal (diff site)

- Risk factors -

- i> Highest Risk of ectopic — H/o Previous Ectopic
- | Previous History $\xrightarrow{\text{↑ Risk by 15\%}}$
- 2 Previous History $\xrightarrow{\text{"}} \xrightarrow{\text{30\%}}$
- ii> 2nd highest Risk — H/o Tubal Surgery
- iii> M/c Risk factor — PID
- iv> cervicitis
- v> Infertility
- vi> ART (gVF)
- vii> Smoking
- viii> Previous C.S
- ix> Contraception & Ectopic
- ↳ All contraceptive ↓ Absolute Risk of Ectopic;
- certain $\frac{\uparrow \text{Relative Risk of Ectopic}}{\downarrow}$
- ↳ Tubal Ligation > IUD > POP
- ↳ Doesn't inhibit ovulation
- so; More Risk of ♀ (ectopic)
- Intrauterine Device
Oral Pill
Boges +
Terane
Oral Pill

order of gvd in descending order of its relative risk
of ectopic ♀ ⇒ Progestasert > Mirena > Cu $\uparrow\downarrow$

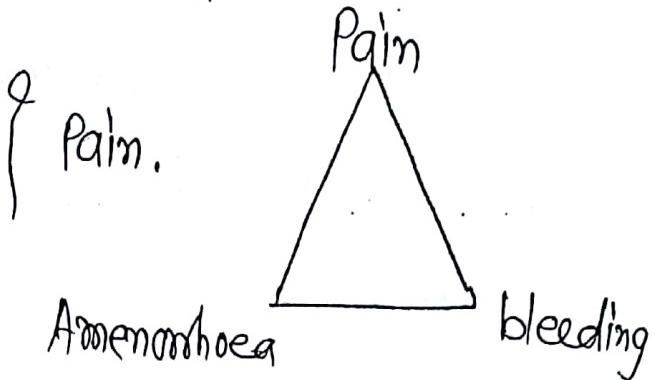
It has gvd & Progestrone both.

- * M/c site of ectopic ⇒ Fallopian tube
 - \uparrow M/c site = Ampulla
 - \downarrow M/c site = Intramural
- * L/c site of ectopic ⇒ C.S. Scal > cervical > Abdominal
 - \uparrow L/c site
 - \downarrow C.S. Scal
 - \downarrow cervical
 - \downarrow Abdominal
- * M/c outcome of Ampullary ectopic ♀ = Tubal Abortion
- * 2nd M/c outcome of Ampullary ectopic ♀ = Rupture @ 8th week
- * M/c outcome of Gestational ectopic ♀ = Rupture @ 6th week
- * M/c time of Gestational ectopic ♀ Rupture = 12th week
 - \uparrow or
 - \downarrow Intramural
 - \uparrow Most life threatening
 - \downarrow Hemoperitoneum ↑↑

* Triad of ectopic ♀ ⇒ Seen in soft patient

Most common

Most consistent



- H/o Nausea; Vomiting
Shoulder tip pain
 - ↳ Hemoperitoneum
 - ↳ Diaphragm
 - ↓
Referred Pain via Phrenic Nerve
- Syncopal Attack
- Pel Abdomen examination ⇒ Tenderness in Lower Abdomen
 - ↳ Rigidity/ Guarding
 - ↳ + Peritonitis
 - ↓
i.e. Ruptured ectopic
- Pel vaginal examination ⇒ Uterus - Enlarged; less perimetrial gestation.
 - Cx Motion tenderness
 - Salpingitis
 - differentiate by Bls ↳ PID → close slg for ectopic.

(65)

Adnexal tenderness

Adnexal Mass QQ

Investigation \Rightarrow UPT \oplus re @ 99% times

Investigation of Choice \Rightarrow Trans vaginal ultrasound

1st finding; which Raises suspicion

↳ empty Uterus

Blinding; which Raises suspicion Against

Not diagnostic
of ectopic

↙
Complex
Lv Adnexal Mass.
↳ Ring of fire sign
on Doppler study

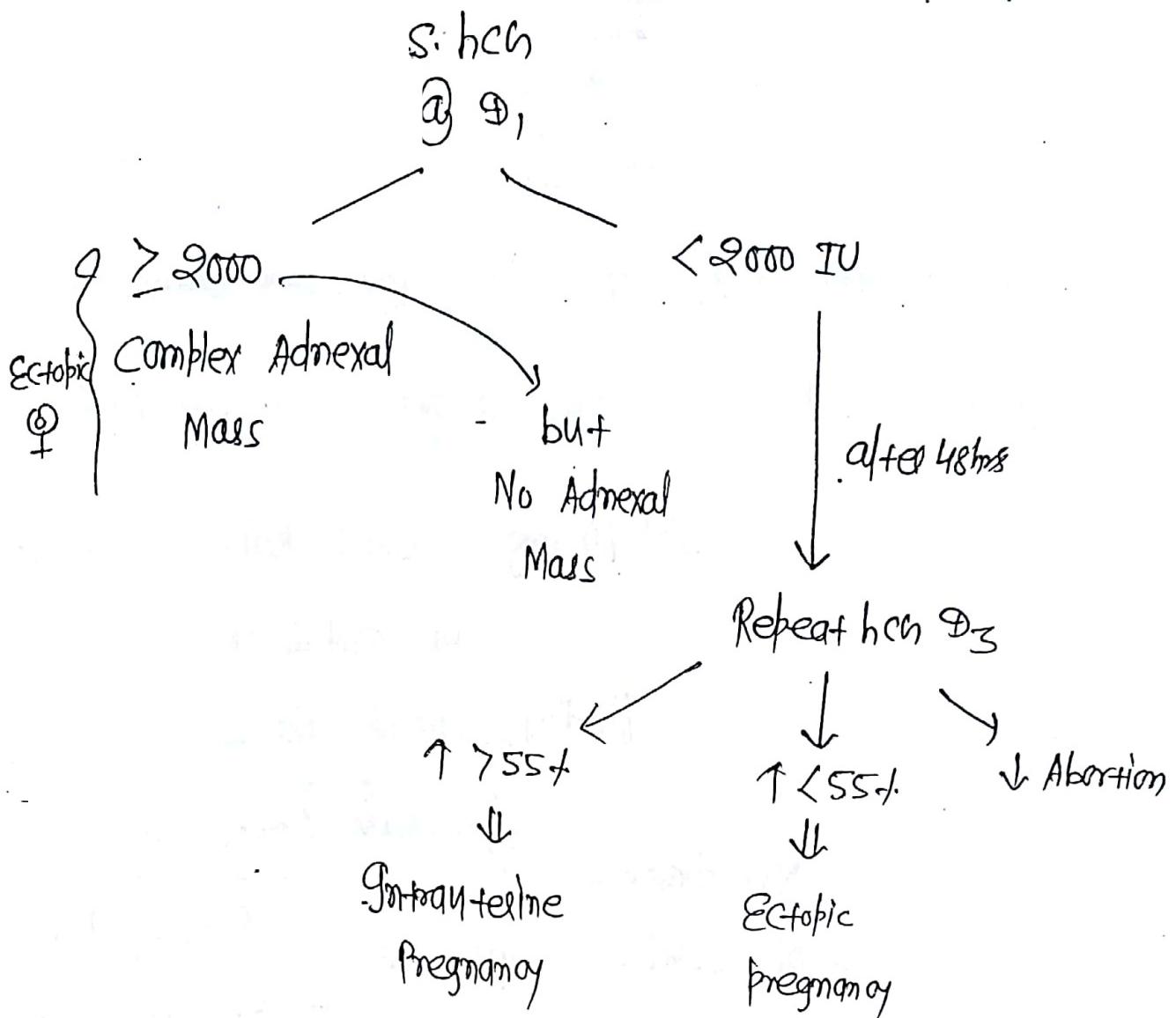
diagnosis finding in ectopic \varnothing = Extra uterine gestation
Sac + cardiac activity.

QQ No Intra Uterine Gestation Sac.; B/L Adnexa (N);

Please correlate clinically.



Pregnancy of Unknown origin; check S. hgs on \varnothing ,



* Repeat hCG till it crosses the critical value

* gOC = TVS + Serial β hCG
 (\leq) $(=)$

* Gold Standard test = Diagnostic Laparoscopy

* Serum Progesterone = $> 25 \text{ ng}$ — Live Intrauterine ♀

$< 5 \text{ ng}$ — Abortion

* culdocentesis
 i) if small collection \Rightarrow OSA;
 ii) if large collection \Rightarrow PTV available

Retroverted Ectopic ($> 100 \text{ ml}$ significant)

ectopic

* Which test has No Role In Ectopic ♀ →

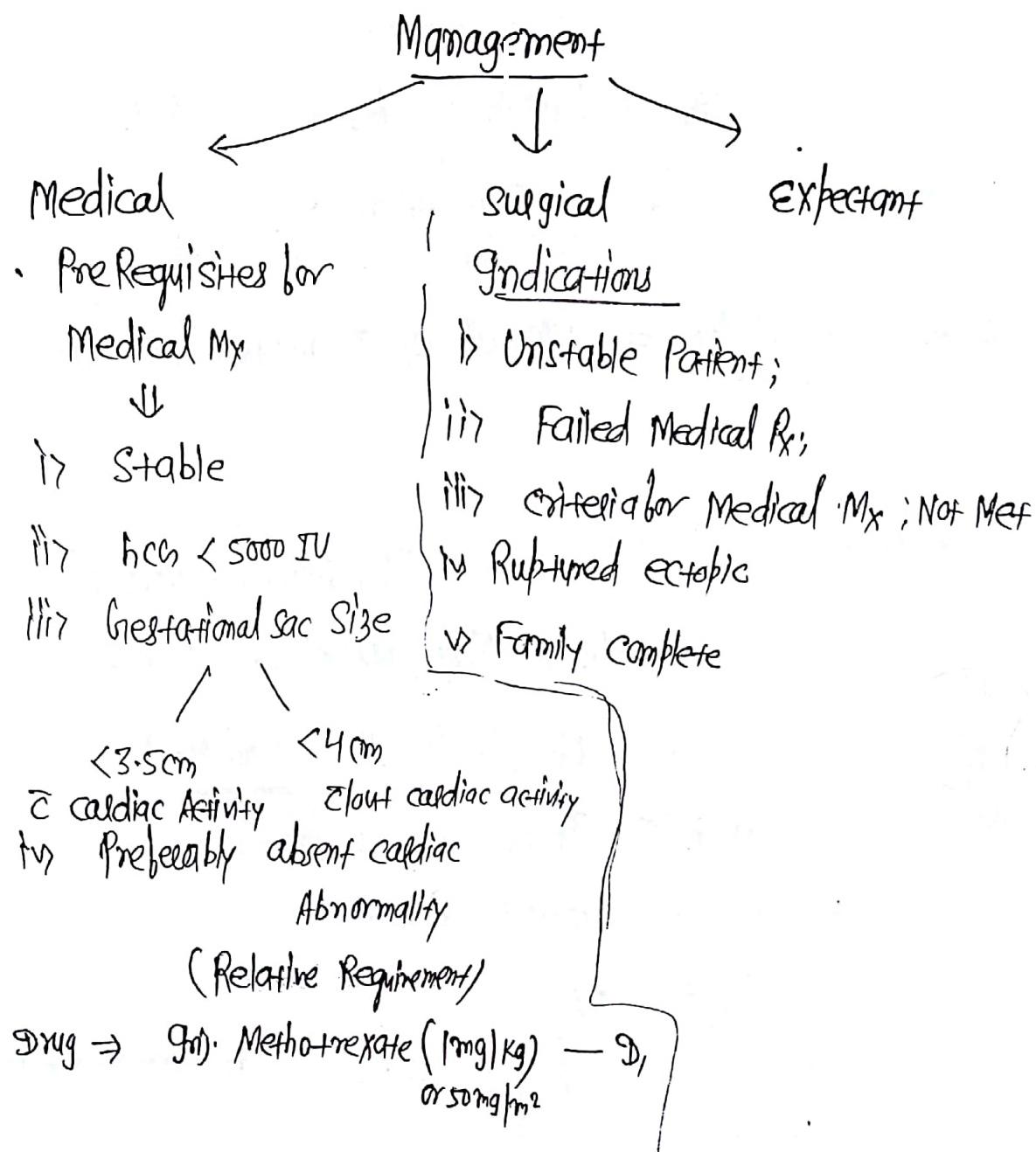
→ Colostomy

118 HSG

Hysteroscopy

{ claim up to date patient

~~is all~~



D_4 S. hct

Management is successful if

D_7 S. hct

S. hct level fall by 15% from D_4 value

if D_7 value fall but less than 15% from D_4 value



give 9mL Methotrexate (D_1)



D_4



D_7 (if again less than 15% of D_4 value)

give 9mL Methotrexate

Failed Medical Mx \Rightarrow Total of 3 Methotrexate Injections

Surgical Mx \hookrightarrow Unstable vitals pt.

Next Most appropriate Step \hookrightarrow

a) i/v fluids & Medical Mx*

b) ~~FAST~~ Immediate Laparotomy

c) Serial B-hct

~~FAST~~

If localizing sign \oplus eg \Rightarrow Peritonitis - do immediate laparotomy

If No localizing sign \ominus - do FAST

I) In general Laparoscopy is preferred over Laparotomy.



(67)

but if Unstable vitals $\oplus \Rightarrow$ go for Laparotomy.

II) Salpingostomy - Is preferred over Salpingectomy



Small Incision (1cm)

on Anti-Megestrin border

(No closure of Incision)

\hookrightarrow b/c chance of ectopic

Ties by suturing; suturing held

by fibrosis & Fibrosis per chance

of ectopic ♀

i) Family complete

ii) Ruptured ectopic

iii) $\geq 5\text{cm}$

iv) If you can't achieve hemostasis

Q.Q. Gyr of Marriage — Infertility — conceived. . . Rupture.
ectopic II

a) Medical Mx

b) Salpingostomy

~~c) Salpingectomy~~

d) Expectant Mx

Partial

Late date

\hookrightarrow Tubal Reanastomosis.

* Expectant Management \Rightarrow

- Pt. is stable
- $\beta\text{-HCG} < 2000 \text{ IU}$ & falling trend
- No visible gestational Sac
- Monitor serial $\beta\text{-hCG}$

* Heterotopic Ectopic \rightarrow

- Risk factor = ART (gVF)
- missed on USG
- \downarrow
- Care diagnosed \Rightarrow by 16 weeks
 - \hookrightarrow Big size Sac
 - \hookrightarrow Impending Rupture / Ruptured

TOC: Medical Mx is c/I;

Sx \Rightarrow Laparoscopic Salpingectomy

If we want to give drug in clinical trials \Rightarrow inj. KCl

under USG guidance \leftarrow Cardiotonic
give to ectopic child & kill.

* Cervical ectopic

- ↳ Painless bleeding
- ↳ Medical Mx (as long as Pt. is stable)
- ↳ Criteria for Dx of cervical ectopic
 - ↳ Parham's (Rubin's criteria)

(68)

* Abdominal ectopic ~~ectopic~~ ⇒ Pain; No Bleeding

- Studdiford criteria

- Late - 3rd trimester
(Abd. ♀)

- 32 week Abd. ♀



↳ Immediate Laparotomy

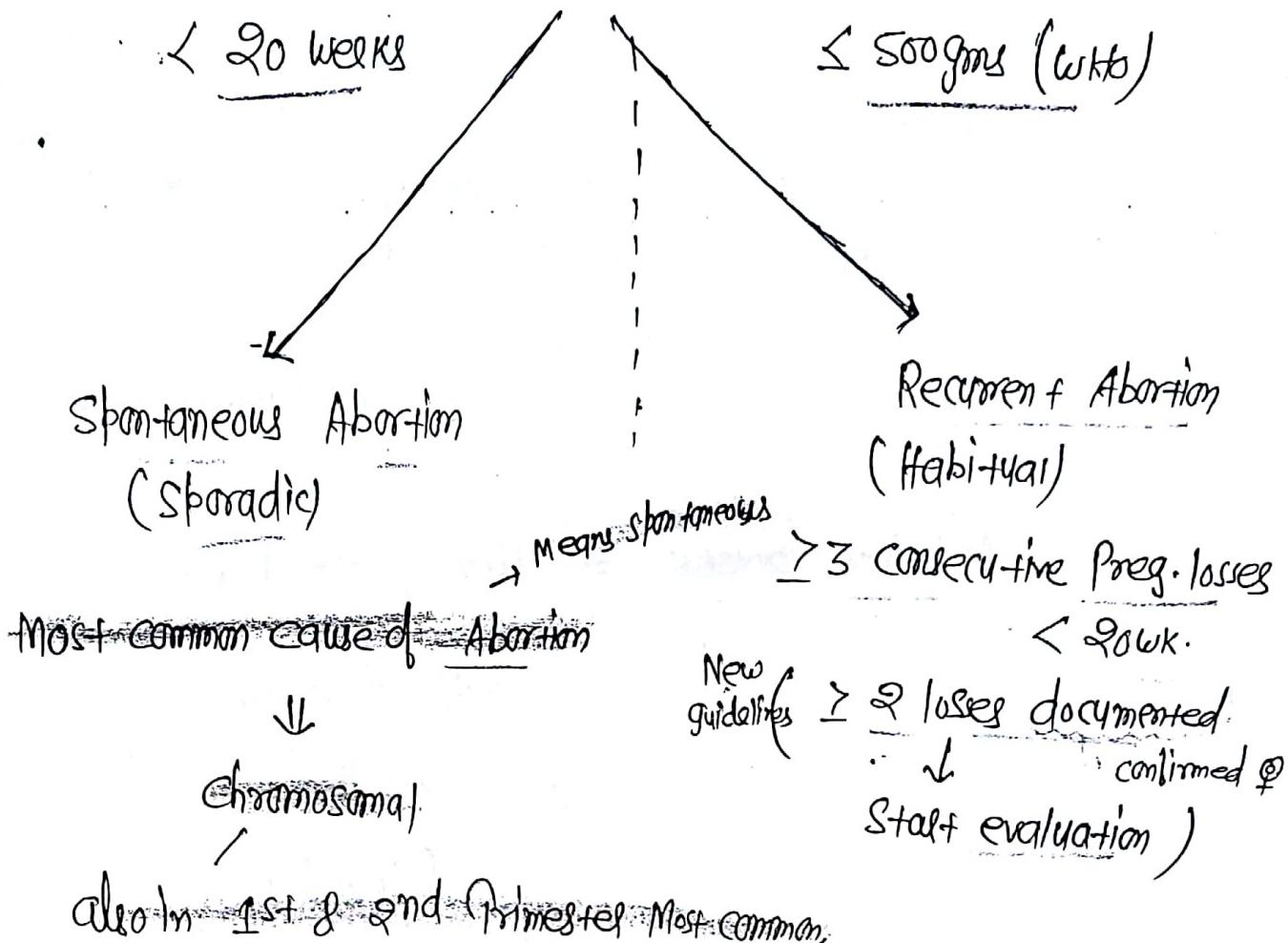
↳ deliver the + Placenta in situ
baby

↳ Leads to Auto-hytic
digestion

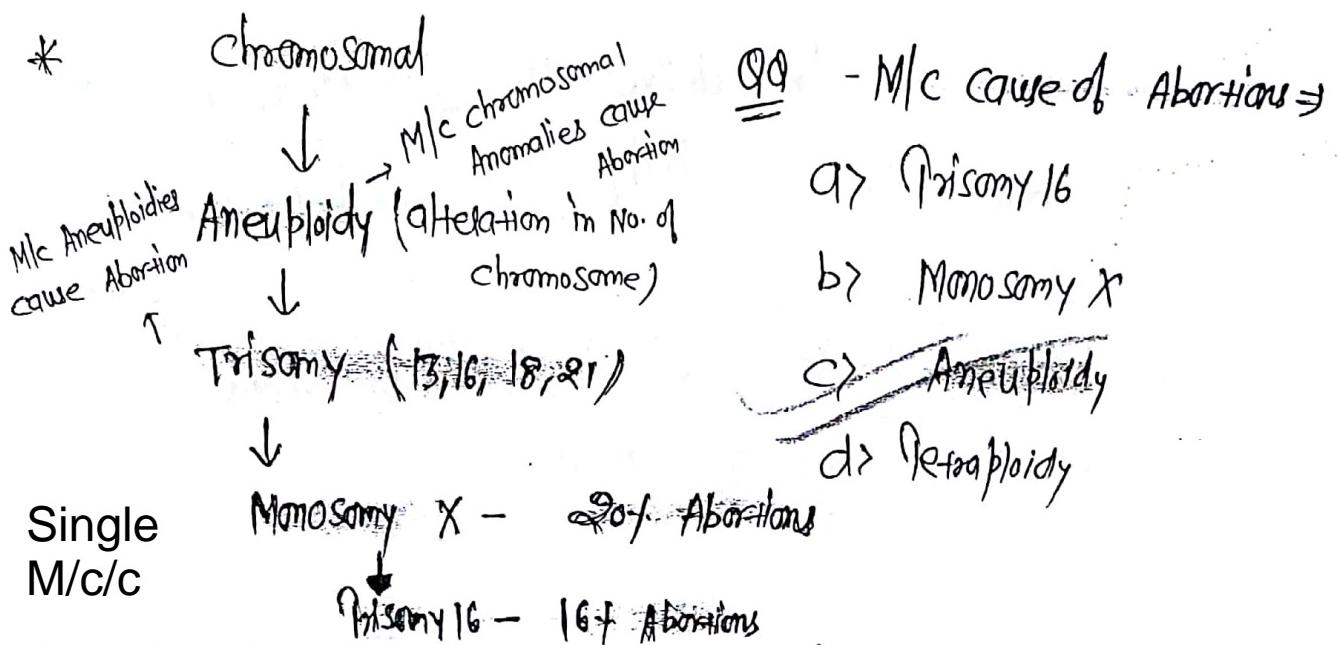
* Ovarian ectopic ⇒ Siegelsberg criteria

↳ Surgical Mx

ABORTION ** weight of baby @ 20 weeks
4 300 gms



* 50% of total abortion in 1st Trimester }
35% of total abortion in 2nd Trimester } Chromosomal



* Most Viable Aneuploidy = Trisomy 21

Most Lethal Aneuploidy = Trisomy 16

(69)

* M/C cause of Recurrent ♀ Loss \Rightarrow Siplopathic (75% cases)
(RPL)

\downarrow
APLA - 16% RPL

\downarrow
Uterine Anomalies
Congenital
Acquired

\downarrow

Endocrinopathies

\downarrow

M/C \Rightarrow Balanced Robertsonian
Translocations

Chromosomal - Only 4% cases of
RPL

* Which of the following doesn't cause RPL?

1) Infection - Syphilis

\Downarrow

TORCH Infections

\rightarrow Can't cause early
RPL

Which Infection can cause RPL \Rightarrow "Syphilis"

\Downarrow \rightarrow Mainly cause

Kasnowitz's Law Still birth,
Not Abortion

if 1st child $\xrightarrow[\text{Loss} @]{\text{Pos}} 1^{\text{st}} \text{trick}$

2nd " $\xrightarrow{\text{"}} 2^{\text{nd}} \text{trick}$

3rd " $\xrightarrow{\text{"}} 3^{\text{rd}} \text{trick}$

4th " $\xrightarrow{\text{"}} \text{Live born child}$
 $\curvearrowleft \text{Stigmata syphilis}$

Every Successive ♀ in Syphilis pos. of Loss
Keeps Testing

Q. G₁ P₀ A₄ (a) 12 weeks present \cong Missed Abortion.
all her previous early losses; all the following Investigatory
for her evaluation except

(1) Karyotyping

(2) LAC

(3) HbA

(4) ~~SIDRL~~

* Karyotyping should be done in patient of RPL

* APLA (Anti Phospholipid Antibody Syndrome) \Rightarrow

↳ Single Most common cause of RPL.

Diagnosis of APLA \Rightarrow 1 clinical + 1 Lab criteria

↳ Any one of the following

↳ i) ≥ 3 ♀ losses of <10wks

ii) ≥ 1 ♀ losses \geq 10wks of a Morphologically
Normal fetus

iii) At least 1 preterm delivery (< 34 wk)

↳ Secondary to Severe Pre-eclampsia
or Hepatic Insufficiency

iv) Venous & arterial thrombosis

Lab criteria \Rightarrow Antibodies

i) LAC \Rightarrow Lupus Anticoagulant \Rightarrow M/c

ii) ACA \Rightarrow Anti cardiolipin Antibodies

iii) Anti β_2 GPI Antibodies \Rightarrow Most Specific

\hookrightarrow Clement disease activity

test to look \Rightarrow Russel viper venom test.
for LAC

IgM & IgG $\overset{\text{ACA}}{\text{+ve}}$ in Medium to high titres on 2 occasions
done 12 wks apart.

* if the pt. is K/c/o APLA syndrome & ♀ also

Heparin

Low M_w Heparin

(Unfractionated can
be used)

\Downarrow

Started when Intrauterine ♀
confirmed

Aspirin

Low dose Aspirin

\Downarrow

Started when UPT +ve

Continued throughout

Hchain stopped

@ onset of Labour

Aspirin stopped

7-10 days before Labour

2nd line \Rightarrow should be used when only 1st line Rx fails.

\Rightarrow Plasmapheresis

Iv Ig

Doc for APLA \Rightarrow ~~Wufalin (Not given in \(\varnothing\))~~
 \downarrow (cause embrofathy)

Doc for APLA in \(\varnothing\) \Rightarrow Low Mw Hefalin.

* Routine test Antibodies for APLA — gm women \rightarrow RPL

* Most characteristic trimester for APLA — 2nd trimester

* UTERINE STRUCTURAL ANOMALIES CAUSES ABORTION

congenital

- Septate Uterus

\downarrow
It can even cause
1st trimester
abortions

Acquired

- ~~Cervical incompetence~~ \rightarrow M/c Acquired
Cancer abortive
- Fibroid (SubMucosal)
- Polyp
- Adhesion

Diagnosis of cervical incompetence

USG based diagnosis

- i) do in 2nd trimester & see cervical length \Rightarrow if it is $< 25\text{mm}$,
- & gestos diameter $> 2\text{cm}$

abnormal in subhili

History based diagnosis

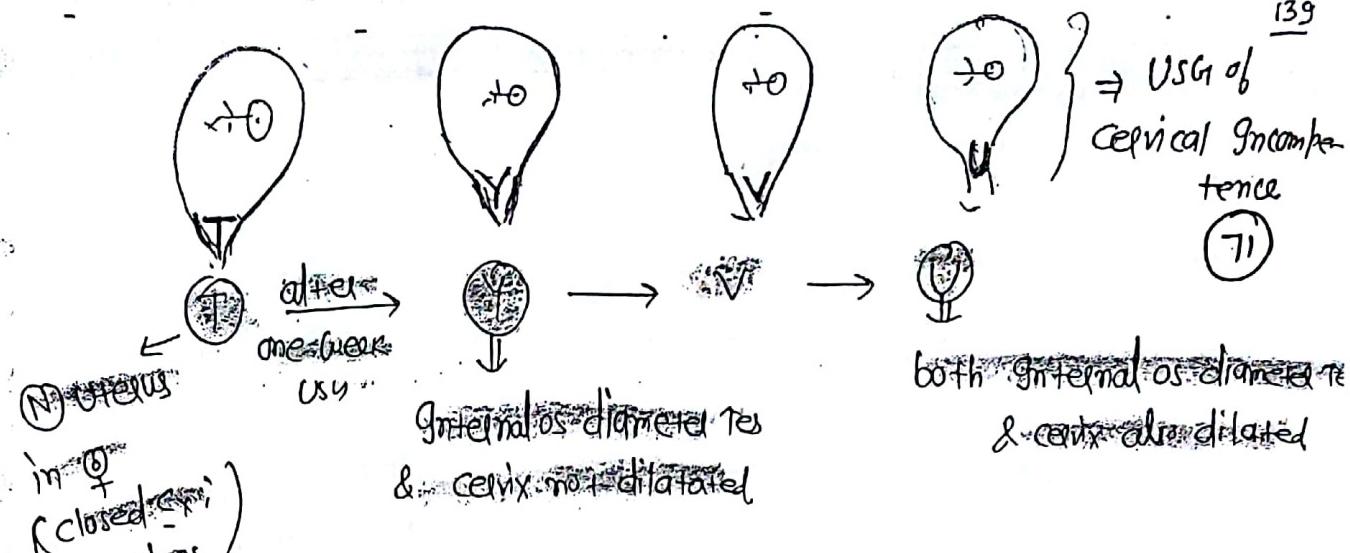
- i) causes 2nd Trimester abortions

(Not causes
1st Trimester
abortion)

in RPL

- ii) painless dilatation of cervix

\hookleftarrow in ovary, fibroids, pregnancy, the placenta less keeps vein,



* Diagnosis of cervical incompetence in Non-pregnancy state :

- Passage of No 8 Hagedorn's dilator through the internal os clout resistance
 - done in premenstrual period
- No 16 Foley catheter → fill the balloon \sim 2cc Normal saline
 - ↓
 - pull it out clout resistance from the internal os
 - ↓
 - Cervical incompetence \oplus

* Screening for Uterine Anomalies (Mullerian Anomalies)

HSG

Sonohysterography

do MRI / Hysteroscopy / Laparoscopy

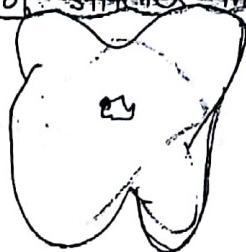
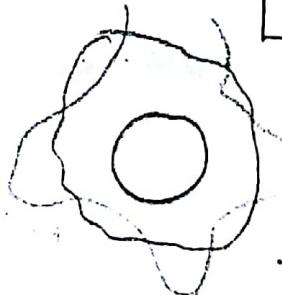
↳ for diagnosis

If Pt. has Cx incompetence ??

do cerclage (cervical stitch)

↳ McDonald Procedure (McDonald)

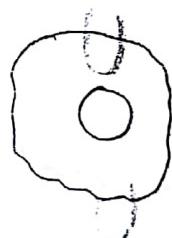
Placement of stitches in the cervix to hold it closed



Purse String Suture

Passes through all the walls of Cx.

Shirodkar cerclage \Rightarrow We Meleglane tape +

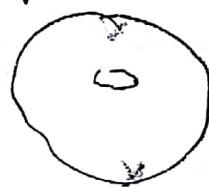


only pass through A-P walls

Cervix Gr index = $\frac{\text{Final Length}}{\text{Endocervical Length}}$

0.32 (N)

> 0.52 \Rightarrow cervical insufficiency



done when McDonald's fail.

* Ideal time to do \Rightarrow 12-14 weeks (14 weeks)

Up to what time we put the cerclage \Rightarrow Up to 24 weeks

At what level we put the cerclage \Rightarrow as close to internal os

Contraindication of cerclage \Rightarrow i) GIC

ii) Current pelvic infection

iii) Rubbed Membrane

iv) Placenta previa

Removal of Suture \Rightarrow @ 37 weeks

SOS if any of the following develop \Rightarrow Ruptured Membrane
↳ Necessary
Chorioamnionitis
Quadrant labor

* In Non-♀ State
Rx of cervical incompetence

⇒



Lash & Lash Surgery

(72)

Q. Should we do Sono hysteroscopy / HSG in Recurrent Pre-term loss

↓

Yes (Should be done as a routine in Non-♀)

In ♀ do USG

↓

ENDOCRINOPATHIES CAUSES RPL

* Thyroid II - Hypothyroidism
do TSH → as Routine Screening for RPL
↳ Subclinical Hypothyroidism → Abortions

* Diabetes → Uncontrolled diabetes
↳ Blood sugar is Not a Routine procedure
↳ do when pt. is Symptomatic / significant family history

* Prolactin → Hyperprolactinemia

S. prolactin → Not a Routine test

Macropathology in ♀ ⇒ Amenorrhea & Infertility

* L.P.D (Luteal Phase defect) → corpus luteum is formed by secreting less Progesterone

↳ Not a established cause of RPL

↳ Only : ~~when Progesterone is low~~

↳ Not a Routine test

* only ~~the~~ established cause of RPL

↳ APLA

Uterine Anomalies

Chromosomal

Hypothyroidism

* How does women \in Abortion presents \Rightarrow

Spontaneous Abortion

Missed

All presents \in

Threatened

Amenorrhea + pain + bleeding

Irreversible

* P/S examination

↳ Os closed

os closed

os open

* Size of uterus

POG

POG

POG

* In UGS examination
Cardiac activity is absent
(missing)

\swarrow
Cardiac activity is
present

Q. Lady is 6 week Amenorrhoeic; Pain + spotting;

USG = Intrauterine gestation Sac & embryo; No cardiac activity.

* i) CRL \geq 7mm & absent cardiac activity (73)

↳ Missed Abortion. ↳ Non-viable pregnancy.

but ii) CRL 5mm & absent cardiac activity

↳ Repeat USG after some times (-Min^m 1 week)

Non-viable ♀ \Rightarrow

○ Mean Sac diameter

$\geq 25\text{ mm}$ & No yolk/embryo
inside

↳ Blighted ovum (No yolk inside b/c of Avascular
(empty sac))

Q. Missed Abortion is Missed b/c Symptoms presented
after later date of Abortion; so; we missed to diagnosis.

Mx \rightarrow Induce Abortion (to open the os)
↳ In Missed Abortion

↳ Empirical Rx (Bed Rest; Progesterone supplements,
1/m-weekly injection; Micronised oral tab)

Avoid intercourse

Avoid Lifting heavy weight

↳ Threatened abortion \Rightarrow abortifacient drugs

No Specific Rx \Rightarrow Inevitable Abortion.

* Complete Abortion

Amenorrhea + Pain + Bleeding + H/o expulsion of foetus
P/S examination

↳ closed



Symptoms improve
after expulsion

USG



For confirmation



Uterine cavity empty

No Retained Product of conception
(RPoC)

* Incomplete Abortion

open

↳ Period of conception
(PoC) can be seen in the
cervical canal

Uterine size full

Bleed a lot

↳ Unstable vitals

(Patient may present

Shock)

Mx
↳ Symptomatic Rx

Mx
↳ Specific

complete the process



do Suction & evacuation
(Digital evacuation could be
life saving)

* Induced Abortion ⇒ MTP Act

Up to 20 weeks

Pt. Asked for Abortion for ⇒ Rape ⇒ Humanitarian

Contraceptive Failure ⇒ Social

~~Ob/Gyn~~ → Eugenic

Medical/surgical disease → Therapeutic

(74)

~~Cohabito~~ Abortion →

RMP's (Registered Medical Practitioner)



- i) degree of dilemma in field of OBG :
- ii) 6 months of leave granted in department of OBG;
- iii) assisted in 25 procedure of abortion in govt. hospital (out of 5 done under demand)

<12 week ⇒ 1 RMP

12-20 week ⇒ 2 RMP

1st Trimester
Up to 7 weeks → OUT patient Medical
Abortion

on D₁ ⇒ Mifepristone (Tab 200 mg oral)

D₃ ⇒ Tab Misoprostol (Tab 800 Mg; Per vaginal;
Per oral; Sublingual;
Buccal)

Usually bleed after Misoprostol
(so; observe 2-3 hr & send home)

D₅ ⇒ History / Examination

of Abortion;
* USG before / After v Not Mandatory

Only in Special circumstance

2nd Trimester
Medical Surgical

M | chy done

Adv: No Anesthesia /
alc to WHO

↳ 800 Mg Sy complication

Disadv: Retained Products
May prf.

Rx

* Prostaglandin

↳ M/c ⇒ Misoprostol
PGE,

2nd M/c ⇒ carboprost
PGE₂

Rx

* D/E

↓

 Dilatation &
Evacuation

Beyond 7 weeks & upto 12 weeks

↳ do "Suction & evacuation"

Hegar's dilator \Rightarrow graduated

blunt + denies (if we perforate)

Medical Rx

Extramammary

Ethacrydine

Intrauterine hyperosmotic

saline

oxytocin

Karman's cannula \Rightarrow 600 mm of negative pressure generated

↳ white plastic device

on P/V examination

↳ Size of cannula = size of uterus

↳ usually 1 less than P/G

it heals itself, wait & watch

Monitor the vitals

↳ if we perforate by Karman's

cannula \Rightarrow immediate laparoscopy

on P/V examination to check

Avascular
Nemesis of
bowel loop

End point of Suction & evacuation

↳ i) bleeding

ii) Air bubbles in cannula

iii) Gripping sensation on cannula

Check cannulae \Rightarrow Sharp devices

↳ give gripping sensations

In village; alternative to Suction & evacuation

\Rightarrow MVA \Rightarrow Manual vacuum aspiration

Form of sponge (sterile)

What to do

Syringe = 60 cc

Pressure $\frac{60 \text{ ml}}{60 \text{ sec}} = 1 \text{ ml/sec}$

Dilatation & Evacuation

(75)

↳ Overm-Forceps \Rightarrow Spoon shaped forceps

No Locks \oplus

Piecelmeal the products

* MOBIUS SYNDROME \Rightarrow ~~Diff. Misprostol~~

↳ Paralysis of Facial Muscle
(Facial Nerve affected)

* If Cx is Not open after giving Misoprostol & all drugs in Undel. 20 weeks

↓

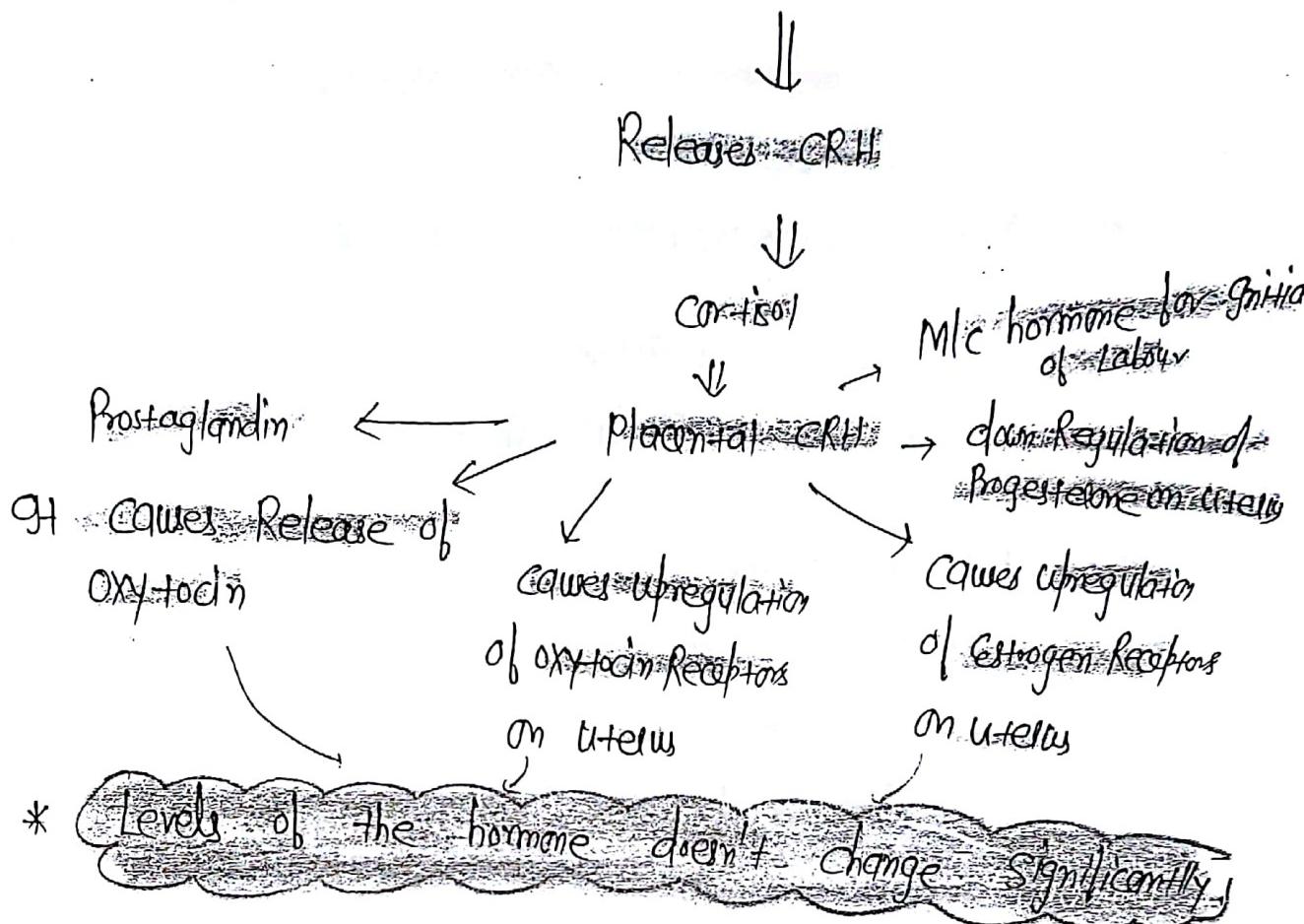
Opening of uterus - In early gestation

↓

Hysterotomy b/c @ 20 wks we don't different ate the lower uterine segment
(So; It gives classical Scal; So; segm after it always do cesarian section in Next ♀)

LABOUR

Initiation of Labour \Rightarrow Functional fetus-hypothalamic-pituitary-axis



Oxytocin comes via \Rightarrow catechol flux

PlFex from the decidua

Maxm Oxytocin Receptors @ 2nd stage of labour

Expected date of delivery = 40 weeks (Naegelss formula)

\hookrightarrow On EDD \Rightarrow 4+ delivery occur

FDD + 1 week \Rightarrow 50% delivery occur

EDD + 2 week \Rightarrow 80% delivery occur

* Preterm labour \Rightarrow before ~~37 weeks~~

Term \Rightarrow Early term \Rightarrow 37 - ~~38 weeks~~

Term \Rightarrow ~~39 - 40 weeks~~

Late term \Rightarrow ~~41 - 42 weeks~~

Post-term \Rightarrow ≥ 42 weeks

* Non-Medically Indicated CS \Rightarrow don't do before ~~37 weeks~~

* Gravida \Rightarrow No. of times the woman has been ♀.

Parity \Rightarrow beyond period of viability.

Abortion (< 20 weeks)

* $G_1 - T - P - A - L$ \hookrightarrow currently Living children.
 Gravida
 term
 $(\geq 37 \text{ weeks})$
 Preterm
 $(20-36 \text{ wk})$

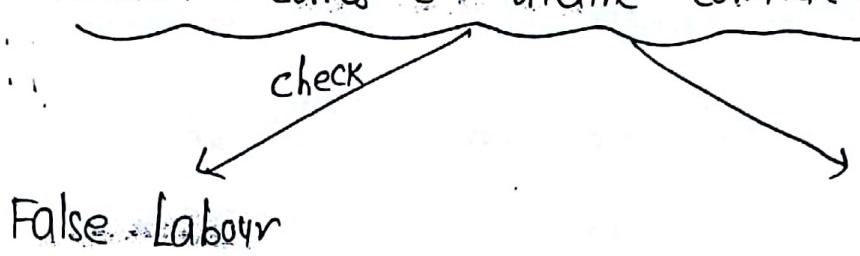
Q: In twin ♀ None of the Parameters change; except

↓
Currently Living children

Q: 2nd Pregnancy; 1st was twins 34 wk delivery; Both alive

$G_2 T_0 P_1 A_0 L_2$

* Patient comes \vec{C} Uterine contraction



False Labour

- Uterine contraction
 \Rightarrow Not ↑ in frequency
 Intensity
 Duration
- No Passage of blood mixed cervical Mucus
- No Progressive ex dilatation & effacement

True Labour

- Uterine contraction \Rightarrow ↑ In frequency
 Intensity
 Duration
- g+ Shows \Rightarrow Passage of blood mixed cervical Mucus
- Progressive ex dilatation & effacement
 - \hookrightarrow g+(cm)
 - \downarrow in.
 - \hookrightarrow Fully dilated
 \hookrightarrow 10 cm

g+ Primigravida \rightarrow effacement \rightarrow dilatation
g+ Multigravida \rightarrow both all simultaneously

- No Rupture of Membrane
- Sedatives \Rightarrow Uterine contraction will subside

- Rupture of Membrane
- Sedatives \rightarrow Pain perception vs Labour will progress

Best Sign \Rightarrow

Progressive
Ex dilatation

Rupture of
Membrane

\hookrightarrow b/c of PROM \Rightarrow Rupture of Membrane before onset of Labour

~~Best to know PROM~~ \Rightarrow Per speculum examination

(15)

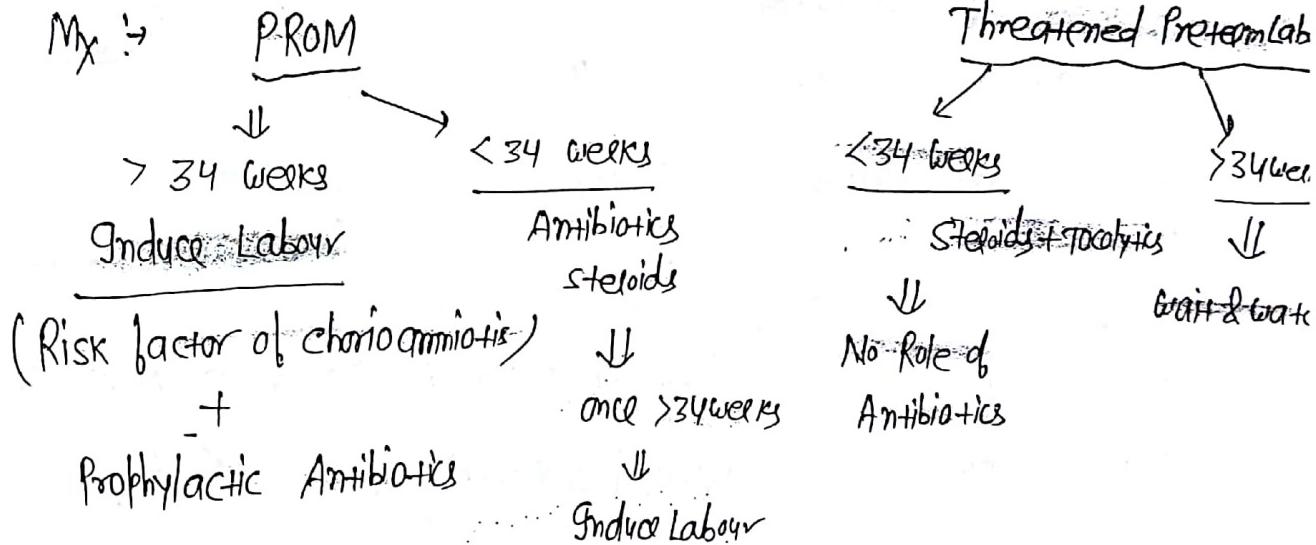
(77)

- Leaking of Fluid from the OS.
- pH \rightarrow Nitrazine paper test
- OSG \rightarrow oligohydramnios
- dye test,
- FFN (Fetal fibronectin)
↳ Marker of Pre-term Labour also

PPROM

\Rightarrow Rupture of Membrane before 37 weeks

Preterm PreMature
Rupture of Membrane



* Most imp. R/F for Pre-term Labour

↳ Previous H/o of Pre-term Labour
~~Infection~~

Prophylaxis in high Risk previous pre-term Labour Patients

↳ Progesterone

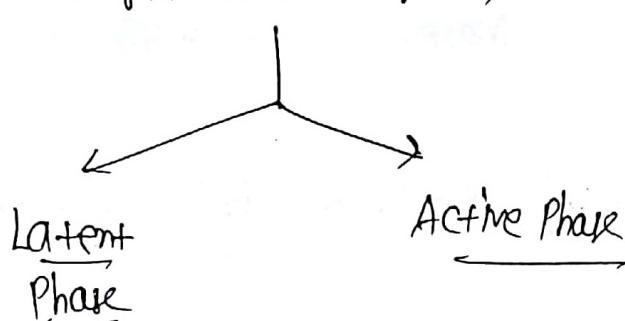
* if patient is in True Labour; we know about? →

1st Stage

Onset of True Labour Pain
to full dilatation of cervix

2nd Stage

From full dilatation to
expulsion of fetus



Cervical dilatation
 $0-5\text{cm}$ ($A(\text{CoGr})$) $\geq 6\text{cm}$

$0-3\text{cm}$ ($C(\text{Ho})$) $\geq 4\text{cm}$

duration of
Latent Phase

| | Normal | Prolonged |
|--------------|--------|-----------|
| Primi | 24 hrs | ≥ 20 hrs |
| Multigravida | 8 hr | ≥ 14 hr |

Rate of cervical dilatation Rate of descent of head
In Primigravida (1.2cm/hr) In Multigravida (1.5cm/hr)

① Progress = 1cm/hr

| | |
|-----------------|-----------------|
| Primigravida | Multigravida |
| 1cm/hr | 2cm/hr |

3rd Stage of Labour ⇒ From the birth of child to complete expulsion of placenta,
4th Stage of Labour ⇒ Duration of observation of Mother : 1 hr

PARTOGRAM

LS3 B,B,B \Rightarrow Blood stained
 M,M \Rightarrow Meconium stained
 C,C,C,C,C

Top Most part \Rightarrow tells about fetus (clear Amniotic fluid,

Middle part \Rightarrow Progress of Labour (Cx dilatation) (78)

Lower part \Rightarrow tell about Mother

tells about

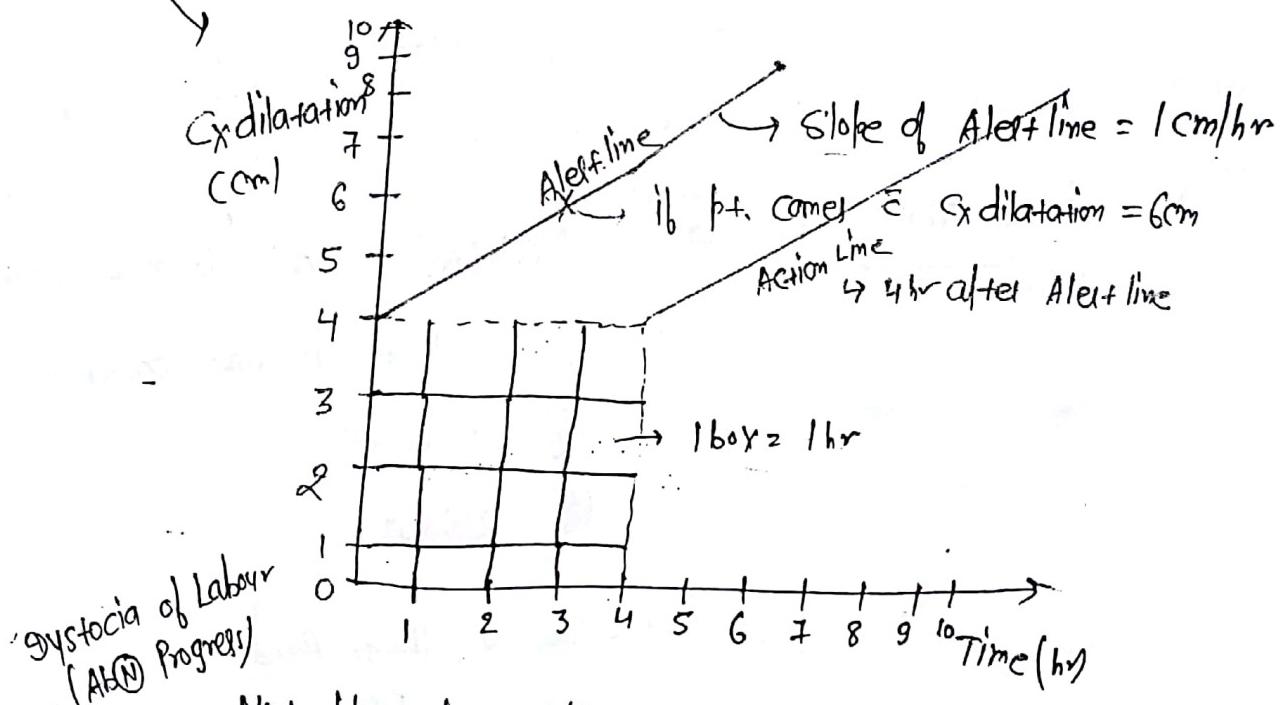


grade 1 \Rightarrow Papilla touch each other

grade 2 \Rightarrow overlaid; can be separated

grade 3 \Rightarrow overlaid; can't be separated

Plotting always done on Alert Line



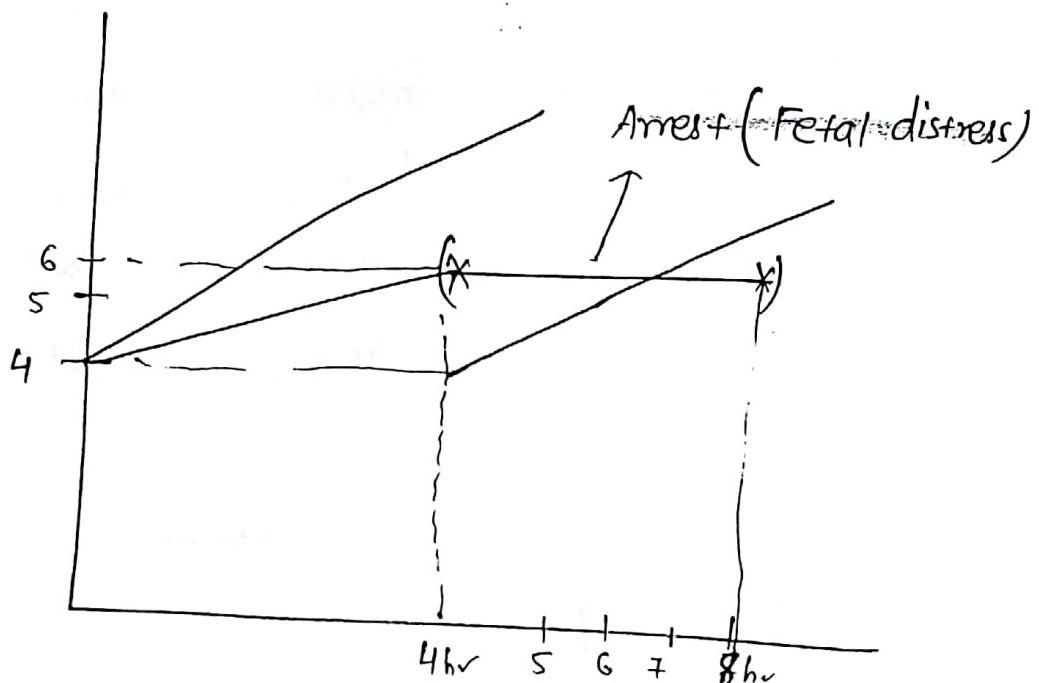
Not plot Latent phase in Partogram (Plot after $\geq 4\text{cm}$)

Partogram is Right to Alert line \Rightarrow Referral to higher center

Partogram goes to Right to Action line \Rightarrow Intervention

Arrest of Active Phase \Rightarrow No change in Cx dilatation even after 4 hrs of Adequate uterine contraction \rightarrow only if FHR $\Rightarrow \text{N}$

Intervention: Cesarean Section



* Lower Most Part \Rightarrow Maternal cond'n (Uterine contraction)

- < 20 sec in one contraction
- 20-40 sec
- > 40 sec

Also tells about \Rightarrow Heart Rate
BP
temp.
Urine output
drugs to be given

2nd Stage of Labour \Rightarrow Avg. duration

\hookrightarrow 1 hr = Primigravida

30 min = Multigravida

Arrest of 2nd stage

\hookrightarrow No descent of head in the presence of Adequate uterine contraction
 for 3 hrs \rightarrow Primigravida
 2 hrs \rightarrow Multigravida

GO G.S.

• Arrest of 2nd stage \in epidural analgesia

4 hr \Rightarrow Primigravida

3 hr \Rightarrow Multigravida

* Progress of Labour \rightarrow P/A \rightarrow Contractions $\begin{cases} \text{duration} \\ \text{frequency} \end{cases}$
 examination

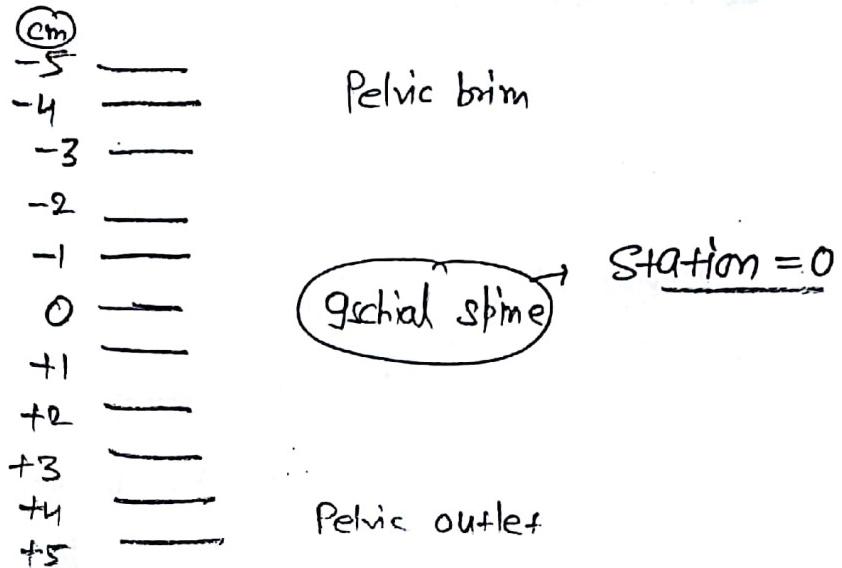
Numelator
 \downarrow
 Parts Palpable

| | Ruled Fine | descent of head |
|-----|--|-----------------|
| 5/5 | A \Rightarrow Entire head above pelvic brim | \Downarrow |
| 4/5 | B \Rightarrow Sinciput high; occiput easily felt | |
| 3/5 | C \Rightarrow Sinciput easily felt; occiput felt | |
| 2/5 | D \Rightarrow Sinciput left; occiput just left | |
| 1/5 | E \Rightarrow Sinciput left; occiput not felt | |
| 0/5 | F \Rightarrow None of head palpable | |

P/V examination \rightarrow C dilatation

\rightarrow Descent of head

Descent of head in p/v examination \Rightarrow



Station -2 \Rightarrow baby's head 2 cm above Gschial spine

Station +3 \Rightarrow baby's head 3 cm below Gschial spine

* LIE \Rightarrow Relationship of Fetus & Long axis of uterus

↓
Firstly correct the dextroversion &

empty the bladder



Longitudinal



Longitudinal



Transverse



Obligate lie =

Unstable lie

↓

Keeps changing even
after 37 weeks

M/c/c \rightarrow Obitotrophic ($> 50\%$)

2nd M/c/c \rightarrow Placenta previa

Polyhydramnios

Oligohydramnios Never cause constable lie

Presentation \Rightarrow Part of fetus which is foremost in the birth canal.

157
⑧



Breech



Cephalic



Shoulder

Leopold Maneuvre \Rightarrow Soft broad part \Rightarrow Buttock
Smooth curve \Rightarrow Back
Limb \Rightarrow Knobby feel

(A) Fundal grip (1st Leopold)

(B) Lateral grip (2nd Leopold)

is with single hand we do

(C) Paralife grip (3rd Leopold) \rightarrow 2nd pelvic grip

(D) Pelvic/suprapelvic (4th Leopold) \rightarrow 1st pelvic grip

grip

Both hands we use

* If head is in complete flexion \Rightarrow vertex presentation

Partial flexion \Rightarrow Simplic presentation

Partial extension \Rightarrow Brow presentation

Complete extension \Rightarrow Face presentation

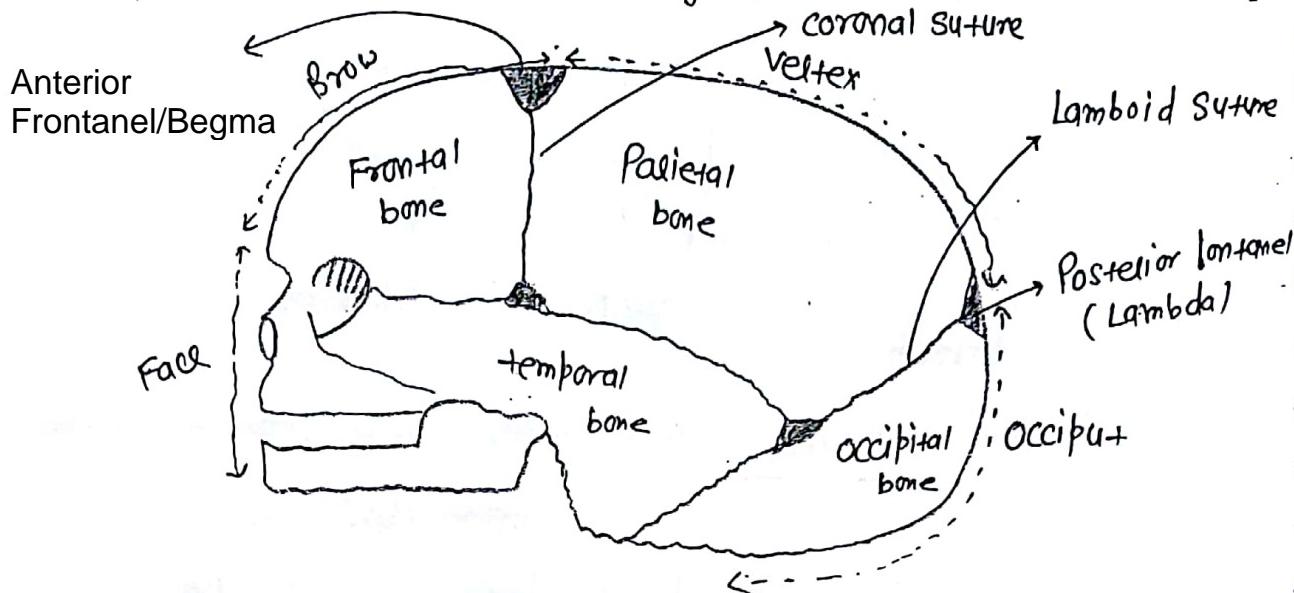
* Anterior fontanel \Rightarrow Bregma

Posterior fontanel \Rightarrow Lambda

Posterior boundary of Brow \Rightarrow Anterior boundary of vertex

* Anterior fontanel \Rightarrow diamond in shape

Posterior fontanel \Rightarrow triangular in shape



Engagement \Rightarrow When the Largest diameter of the presentation crosses the Pelvic brim.

Largest Transverse diameter \Rightarrow Biparietal diameter
L 9.5cm

engaging dm in different presentation \Rightarrow

Engaging diameter (AP)
↳ Antero-
Posterior

Vertex
Suboccipito
bregmatic (9.5cm)
Sinciput (Posterior flexion/ deflection)
Occipito
frontal
(occiput to Anterior
end of Anterior fontanel)
11.5 cm
Sub-occipito frontal
(10.5 cm)

complete expiratory
I - Face
Brow
SubMento-
bregmatic (9.5cm)
Mentoucilli-
ca (14cm)
II
Largest dm
of fetal head

* In Most of the Primigravida

↳ engagement @ 37 weeks

* free floating head @ 37 weeks in Primigravida

↳ diff deflected head

CPD;

Placenta previa;

Polyhydramnios.

cephalo pelvic disproportion at
(CCPD)

* Engagement Rule out cephalo pelvic disproportion at the outlet.

* when the head is engaged; station ≤ 0 .

↳ on P/A exam; Q/S is palpable

* DENOMINATOR - Bony point on the presentation used to describe the position of head

Presentation

Vertex



Denominator

Occiput

Breech



Sacrum

Brow



Frontal bone

Face

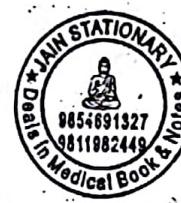


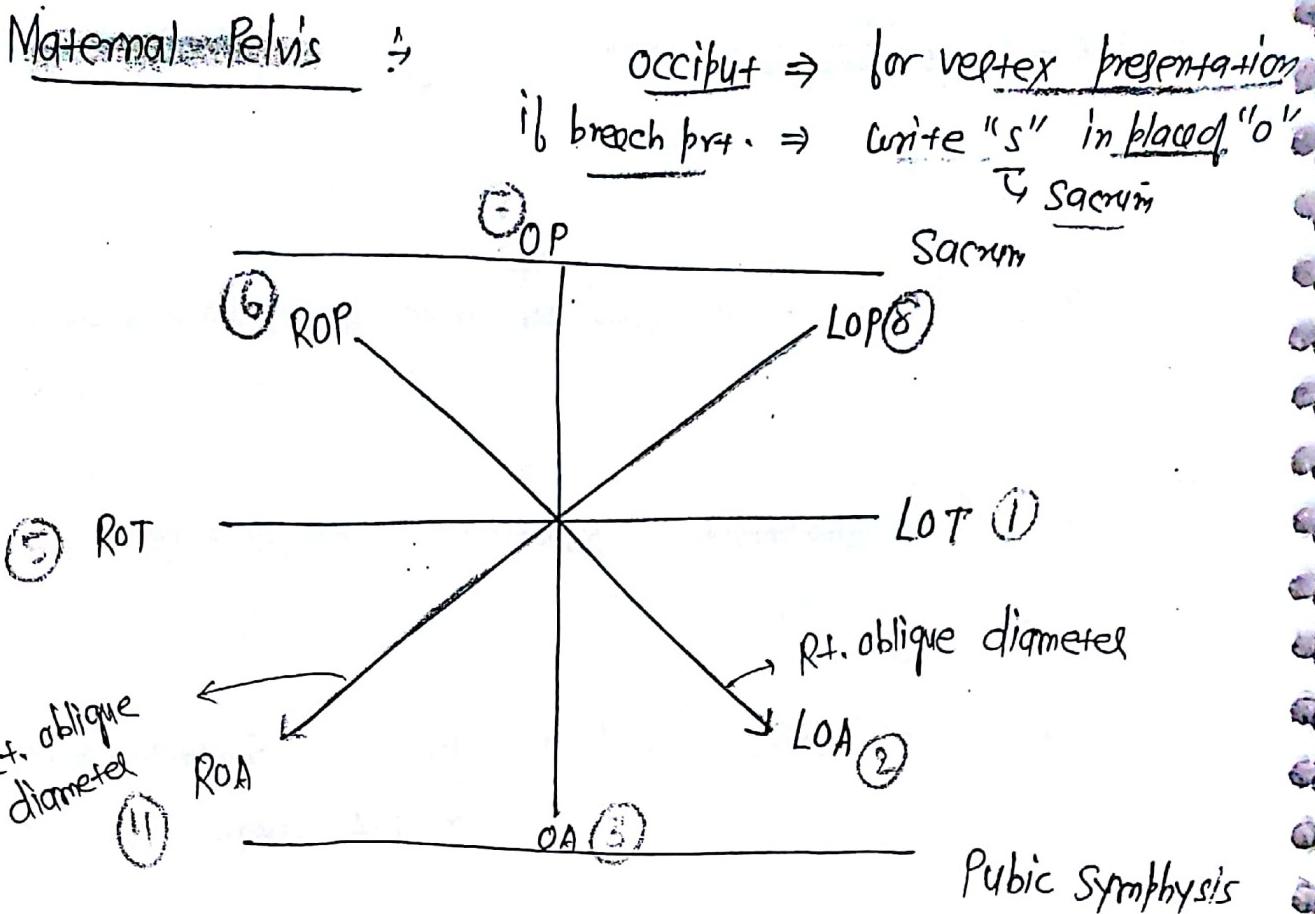
Mentum

Shoulder



Acromian process (Scapula)



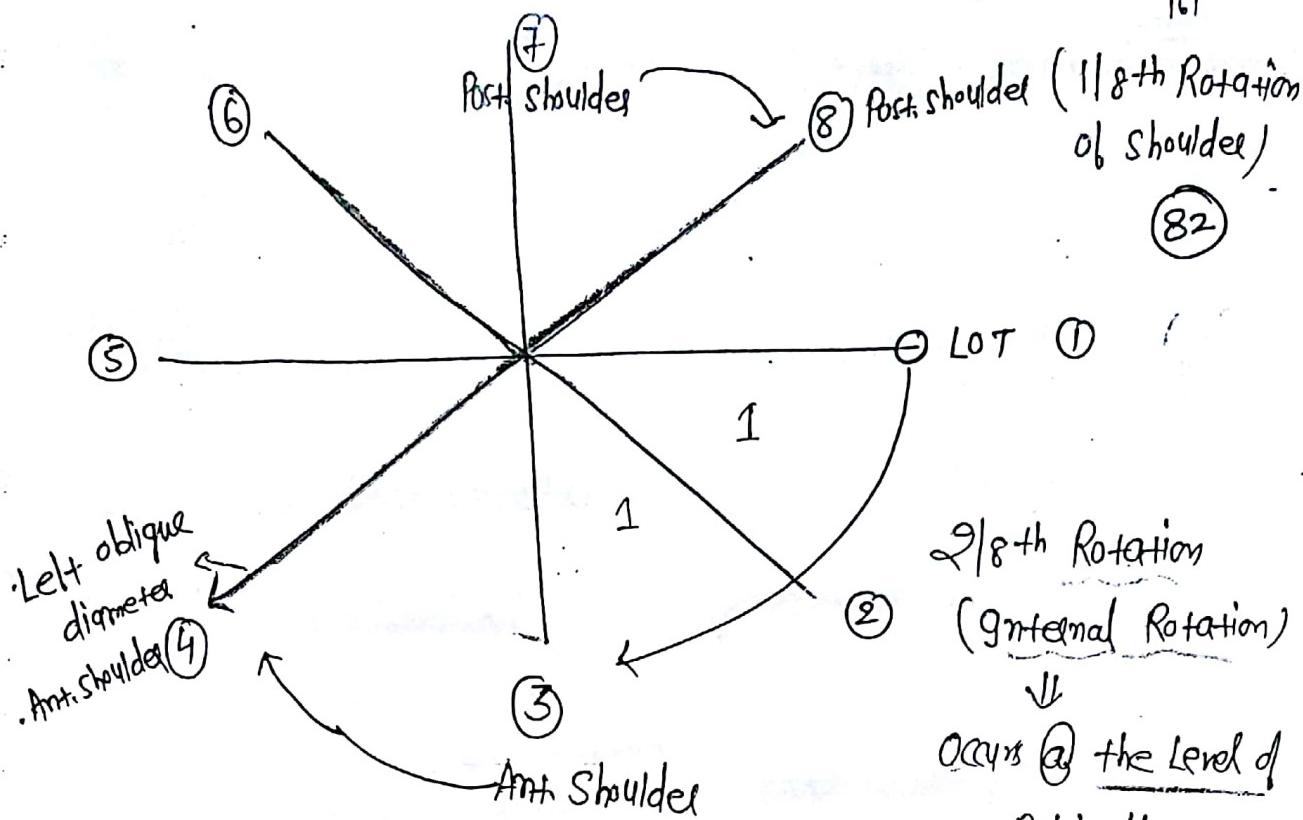


* In Routine exam: Plv exam done in active Labour
4 hourly.

* M/c position of N Labour \Rightarrow LOT**

Cardinal Steps of N Labour

- (1) Engagement
- (2) descent
- (3) flexion
- (4) Internal Rotation
- (5) Extension \Rightarrow delivery of head (occiput -> vertex -> forehead)
(chin -> mouth -> glabella)
- (6) External Rotation

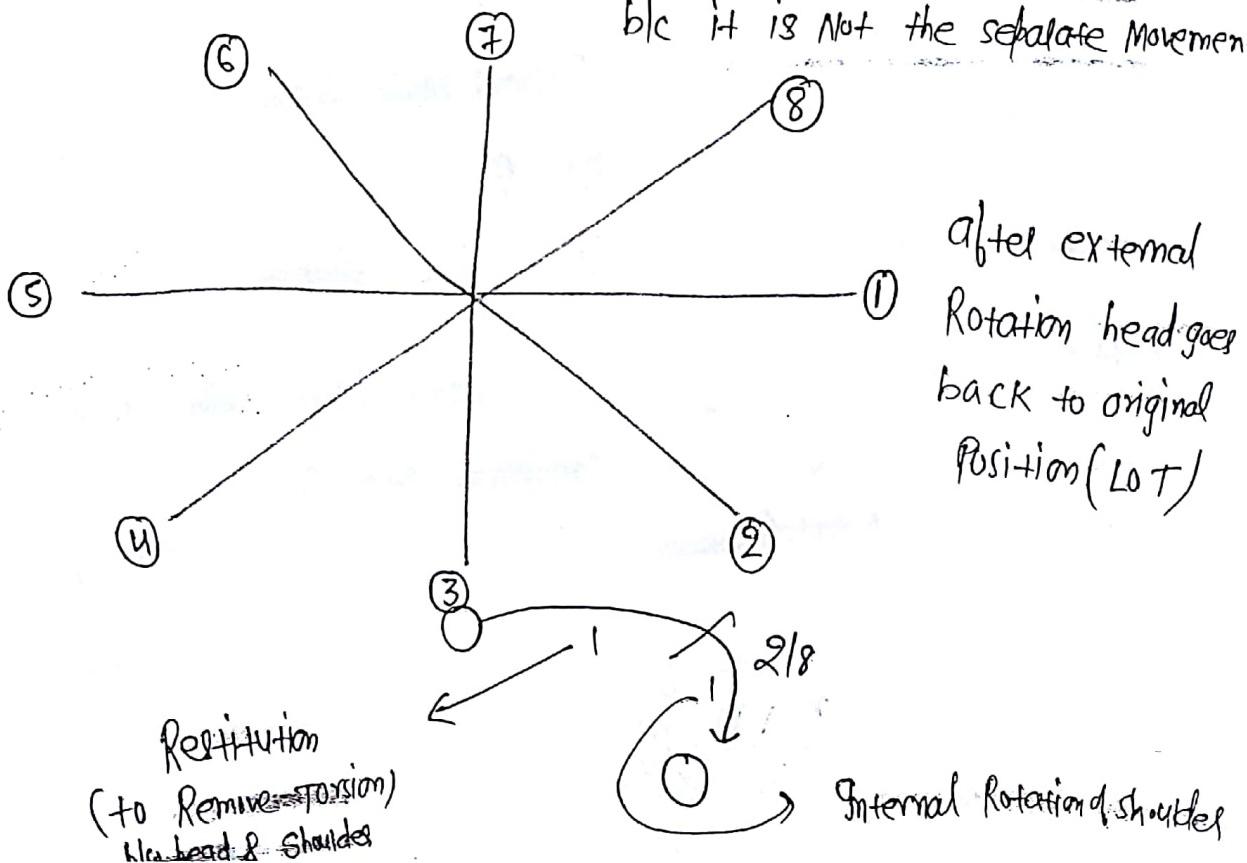


* Relation of Internal Rotation to External Rotation

* Relation of Restitution to External Rotation
↳ opposite direction,
↳ same direction

but Shoulder Rotates only
by $1/8$

* External Rotation ↳ Restitution is Not a cardinal Movement
b/c it is Not the separate Movement



3 P's Required for Labour \rightarrow Passage

Push

Passenger

Passage \Rightarrow

Maternal Pelvis \Rightarrow by ~~calipers~~ \downarrow ~~on the bony border~~

M/c Pelvis in ♀ = Gynaecoid Pelvis (circular Pelvis)



$4 \frac{1}{2} \text{ in } 50 \text{ cm}$

2nd M/c Pelvis in ♀

Transverse diameter \geq AP diameter
= Anthropoid Pelvis



Antero-posteriorly oval Pelvis

in ♂ & ♀

\bigcirc AP diameter
 ∇ Transverse diameter

Other = Android Pelvis



Typical Male Pelvis

20% ♀



Transverse diameter
 ∇ AP diameter

Inlet = Heart shaped

Least common Pelvis in ♀ = 5% Cases (Platybelloid Pelvis)

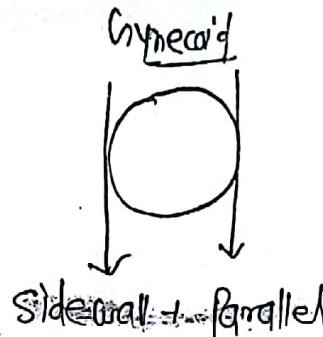


Transversely Oval Inlet

Flat Gynaecoid

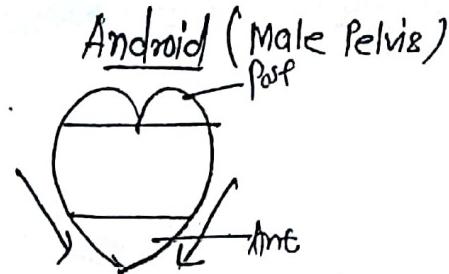


Transverse diameter \ggg AP diameter



Obstetrical spine \Rightarrow Blunt

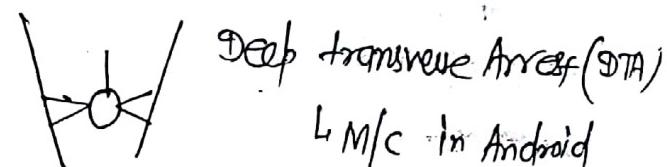
- DTA Not Seen



(83)

M/c of Occipito Posterior \Rightarrow Android Position
(Posterior space is More than Anterior)

Sharp



Subpubic Angle \Rightarrow obtuse
($90-100^\circ$)

Acute
(85°)

Shallow Pelvis

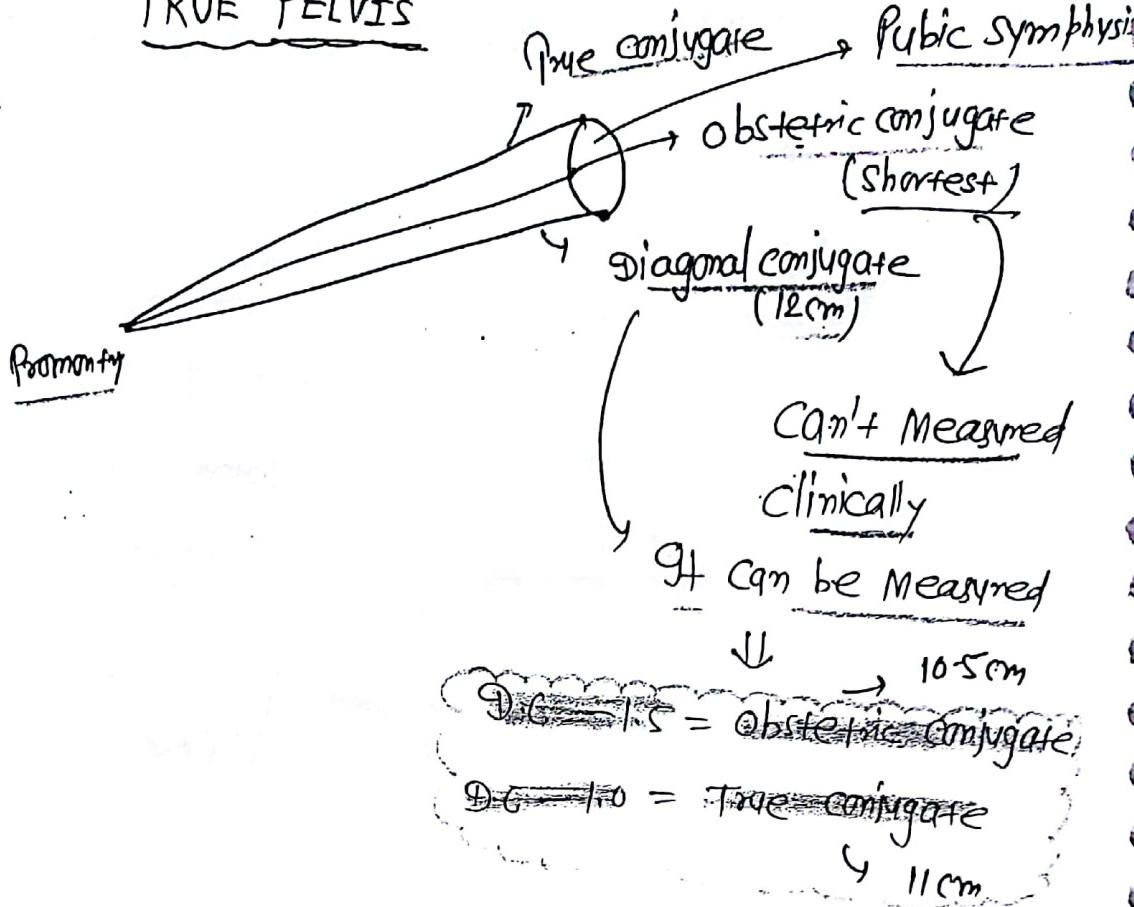
Delt Pelvis

(* Deepest Pelvis \Rightarrow Anthropoid)

occipito posterior \Rightarrow Android
Resistant occipito posterior \Rightarrow Anthropoid
Direct occipito posterior

TRUE PELVIS

INLET →



Cavity:

Inter-Gschiāl spine diameter (IGS) → distance b/w gschiāl spine

↳ 10.5 cm

↳ We Shouldn't Able to touch the both
gschiāl spine simultaneously Normally

Contracted cavity: IGS < 8 cm

OUTLET →

Inter-tuberosity diameter = 11 cm
(gschiāl tuberosity)

↳ "Four-knuckle test" is done to measure it.

Contracted Pelvis \Rightarrow Any 1 or More of the Previously Studied diameters are contracted

(84)

Inlet \Rightarrow $< 10\text{cm}$ (for diagonal conjugate if it is less than 11.5)

Cavity \Rightarrow $< 8\text{cm}$

Outlet \Rightarrow $< 8\text{cm}$

do C.S.

To know about the contracted Pelvis; we will do "Pelvic Assessment"

In Primigravida; it do @ 37 weeks

In Multigravida; it do; when she goes in Labour

Q. G₂P₁; Previous 1 C.S; done for contracted Pelvis; comes
 @ 37 week ANC \Rightarrow Recurrent Indication for C.S.
 (Never do VBAC)

* Cephalo-Pelvic Disproportion (C.P.D.) \Rightarrow

Best way to know \Rightarrow Trial of Labour



MRI



CT Scan



X-Ray Pelvimetry



Pelvic Assessment

G₂P₁L₁; Previous 1 CS done for CPD; she presents c
Labour & Pain 37wk; OS 1cm dilate; 70% effaced vertex
A+ = -3; what is M_X?

do VBAE
 \downarrow

Trial for VBAE

CPD is Non-Recurrent Indication for CS

Forms Roots of obs. outlet
 \uparrow

* Plane of Least Pelvic dimensions = obstetric outlet

= ischial spine
(Passes laterally to ischial spine)

* Anatomic outlet = ischial tuberosities
(Passes laterally to ischial tuberosity)

* Plane of Greatest Pelvic dimensions = disc space b/w S₂-S₃

II. PUSH

Uterine contraction; Pacemaker \Rightarrow R. corny

Strongest \Rightarrow Fundus

Rate of spread \Rightarrow 2 cm/sec; depolarise entire uterus in 15 sec

at what Intrauterine pressure
contraction became palpable

\Rightarrow 10 mm Hg

at what GUP contraction became
Painful

\Rightarrow 15 mm Hg

\hookrightarrow Minⁿ Pressure Required to Initiate
dilatation

* Fundus can't be Intended \Rightarrow Moderate contraction

\Downarrow
40 mm Hg

(85)

* end of 1st Stage \Rightarrow 50 mm Hg
2nd stage \Rightarrow 80 mm Hg

* Adequate Uterine contraction \Rightarrow

- i) 3 contraction in a span of 10 min & each is lasting for 45 sec;
- ii) If contraction generates pressure of 200-250 Montevideo Unit,

Montevideo Units \Rightarrow No. of contraction in 10 min
↓ P' generated

good or Bad?
Bad

Tachysystole \Rightarrow > 5 contractions / 10 min

UPI \rightarrow Fetal distress

Tachysystole + Fetal distress Hypersimulation

b/c Most of blood supply goes to baby in diastole

Rx

- 1^{stly} Stop the infusion
- Left Lateral Posture
- O₂ by Mask
- I/v. fluids
- TOCOLYSIS (Stop the contraction)

* On Augmentation of Labour

Oxytocin given

Misoprostol avoid

↳ May cause hyperstimulation

Rupture uterus

Management of

⇒ LABOUR

FALSE

No change in Cx dilatation



Mx ⇒ Rest & Sedation

TRUE

Progressive Cx dilatation

What stage

Amount of Cx
dilatation

Latent Phase

0-5 cm

(WHO)

Active Phase

> 6 cm

Rest & Sedation

wait & watch

Is the Labour slow?

Yes (<1cm/hr)

No (fast)

Contraction are Adequate

or No+

Yes → Inadequate Labour

No/Adequate

↓
d/t CPD
of Position

Moulding
cervix (swelling
& scalloping)

↓
do Repeat sp. maneuver

↓
In Stove labour
↓
4 Marked
(CPD),

Mx ⇒ Augmentation

ARM (relative Postaglandin)
↓ after min of 30 min Oxytoxin

Q. Pt. comes @ 2 PM. 6 cm dilated Cx & soft effaced;

Vertex @ -1; Membrane absent, Liquor clear, contractile.

220 mV (Montevideo Units) ^(Ruptured)

(86)



@ 6 PM; 7 cm soft effaced; Vx @ -1; caput + ();

Moulding (++)



~~do. Trial of Labour~~

@ 10 PM; Finding absolutely same

↳ i.e. Arrest of Labour

↳ do. G.S.

OBSTRUCTED LABOUR

In General Physical examination \Rightarrow Excessive

dehydrated

Tachycardia

Tachypnoea

Acidotic Breath

On P/A examination \Rightarrow Upper segment \Rightarrow Tonically ~~tonically~~ contracted

Lower Uterine segment = stretched / thinned out

Blew US & LUS = depression (Ring)

Band's Ring / Pathological
Retraction R.

Suprabitis bulge (Bladder)

FHR =

~~Second distress~~

~~Absent~~

P/V examination \Rightarrow

~~Soft/hard~~ ~~engorged~~

* If we put Folay's we can not
be able to enter as it is

~~soft/moulding~~

Compressed by head we can
Only able to put feeding tube
8 can see hematuria

~~Hematuria~~

Doesn't Pass Urine

is the earliest Marker of obstructed
Labour

* obstruction is the absolute Indication for CS, even if the
fetus is dead

* Destructive Procedures \rightarrow Not in Modern Obstetrics

↳ do craniotomy

Symphiotomy

q) Not done CS \Rightarrow Rupture uterus

* if patient present ∞ Not passing urine and told us about bladder
injury then we prove it as not bladder injury by

↳ VVF (vesicovaginal Fictula) in developing countries

↳ obstructed Labour

In developed countries:

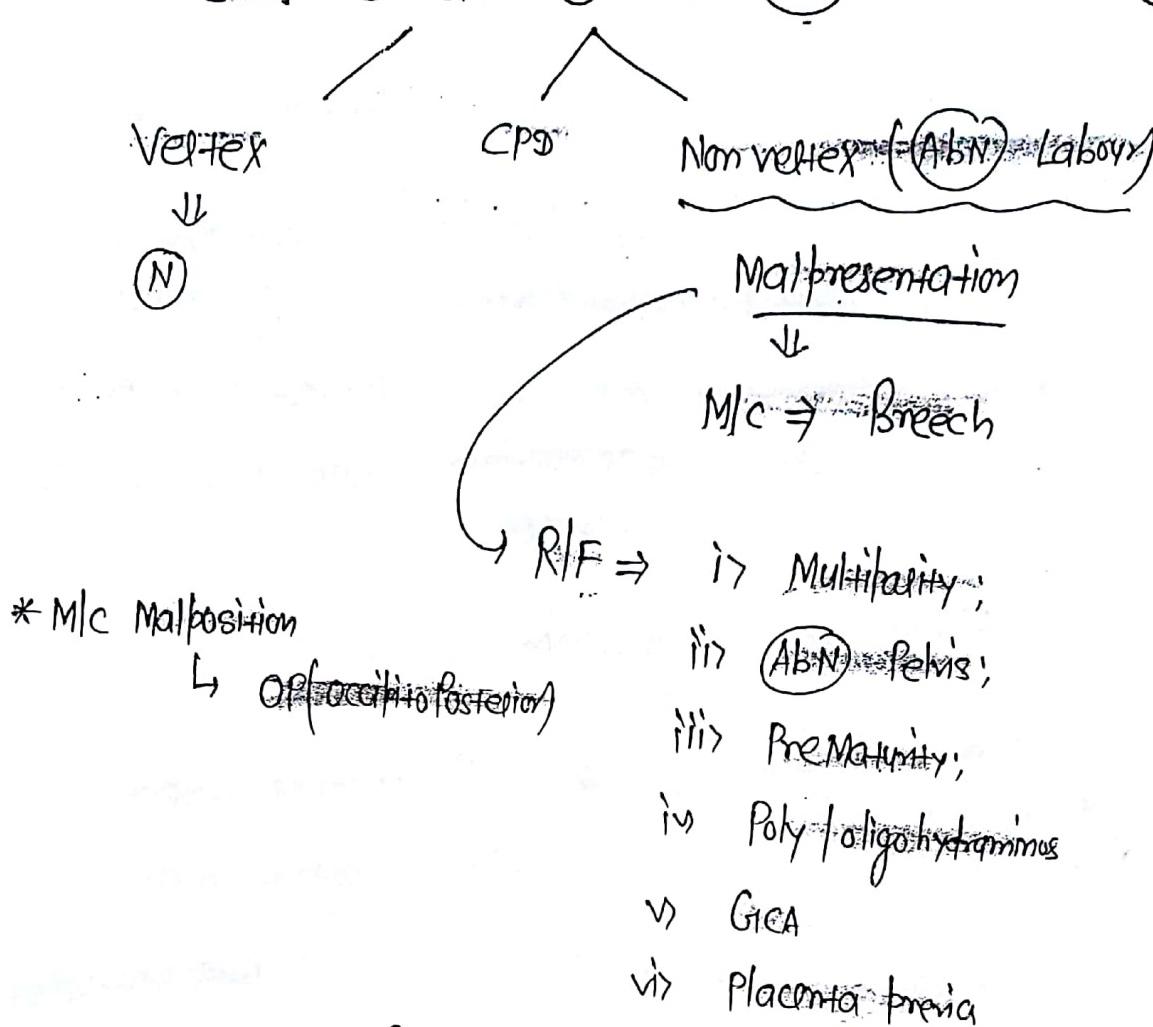
↳ Gynaecology (Malignancy
suspected)

III: Passenger

171

Feature - Convert \textcircled{N} into (\textcircled{ABN}) Labour

87



BREECH

M/c cause \Rightarrow Prematurity

| <u>POG</u> | <u>Breech</u> |
|------------|---------------|
| 28wk | 25+ |
| 32wk | 27+ |
| Term | 34+ |

M/c type of breech \Rightarrow Frank breech (extended breech)

Hip joint flexed, knee joint extended
~~Plumb line doesn't reach heel~~

Complete breech (Flexed breech) \Rightarrow Less common type of breech.

Both knee & hip are flexed; P/v \Rightarrow Ischial tuberosity; Anal opening; Heel pads



* Primigravida \Rightarrow Frank breech

Multigravida \Rightarrow Complete breech

* Footling breech \Rightarrow Incomplete breech

↳ baby May have cord prolapse
P/V \Rightarrow can see foot of baby

Cord prolapse \Rightarrow exposed to temp.

highest risk in Footling breech

↳ intense cord vasospasm

Low risk in Frank breech

↳ death of baby

* Generalization of CS in breech \Rightarrow

Absolute Indication

\Rightarrow ① Footling breech;

② Shoulder breech;

↳ Hyperextended head

Relative Indication of CS.

↳ Vaginal is not C/S, but CS is preferred over vaginal delivery.

① Primigravida \Rightarrow Breech;

② Previous CS \Rightarrow Breech;

③ Macrosomia \Rightarrow Breech;

④ Hydrocephalus

↳ do ventriculo-peritoneal shunt by Pediatric surgeon.

⑤ Prematurity

Q. Primigravida; 37 week; ANC checkup; O/F = Breech presentation; FHR N; Liq N; Placental fundal; Pelvis adequate; Mx = II

(88)

do ECV (> External cephalic version)
 can be done
 In Latent phase of Labour

↳ for all Non-Cephalic presentation.

if Requirements Met.

done for (a) Single term ♀;

(b) ≥ 37 wk / 36 wk baby;

(c) Liquor Adequate;

(d) Membrane should be intact;

(e) FHR N

(f) No C/S for vaginal delivery,

Relative risk for ECV \Rightarrow Risk More than Benefit

↳ avoid ECV \Rightarrow (a) Previous C/S

(b) GUGR

(c) Pre-eclampsia

* ECV is always done under continuous fetal monitoring;

* It is done under tocodynamometer (telestethesio/m);

* during ECV \rightarrow if fetal distress \oplus

↓
 Return baby to original position
 ↳ do C/S.

Q. G₂P₁, L 39 weeks Breech presentation



first try ECV



but if any c/I of ECV ()

→ Primigravida \Rightarrow C-S

→ Multigravida \Rightarrow vaginal delivery

(Assisted) Breech vaginal delivery

→ if fetus has extended Arms \Rightarrow Lowset Maneuver

extended Legs \Rightarrow Pinard's Maneuver

After coming head \Rightarrow Burn-Matthew



Grab foot &
goes 180° towards
Anterior
Pelvis of Mom;
after head comes out
by

Spine of fetus is
& towards
the obstetricians

Maneuver

In this delivery of head by
"Flexion" Not by extension.

After coming head \Rightarrow Mauriceau-Smellie

viet (MSV)



* Burn Matthe & MSV

only when baby is



DorsoAnterior

do Malleolone flexion
& shoulder traction &
delivery of head by "flexion"

In dorso-posterior breech; after coming head \Rightarrow Praguet Maneuver

* Dührssen's Incision \hookrightarrow 2 Small Incision on Cr



\hookrightarrow done in preterm after coming head,
 \hookrightarrow if Most of the part of baby is delivered & only head is inside

* Forceps delivery \Rightarrow Pipers Maneuver

* Last Resort \Rightarrow Zavanelli Maneuver

* M/c cause of Fetal death \Rightarrow cerebral Hemorrhage
 In breech (ICH)

* O.P. (occipito Posterior) \Rightarrow No + Malpresentation; Malposition

\hookrightarrow M/c cause \Rightarrow Android Pelvis

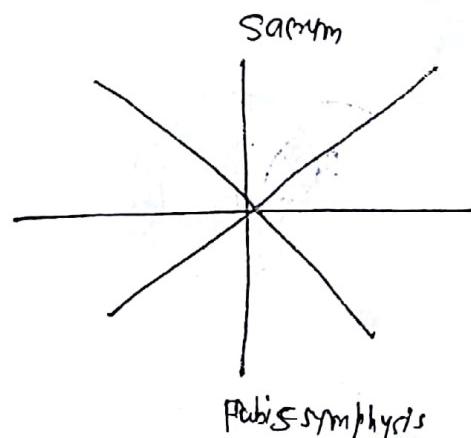
2nd M/c cause \Rightarrow Deferred head

\Downarrow
 Engaged diameter \Rightarrow occipito/mental

410.5

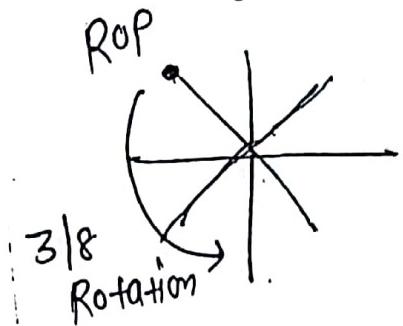
M/c In Primigravida

M/c Position - R.O.P.



1st Outcome \Rightarrow all favourable

- Push
- Passenger
- Passage

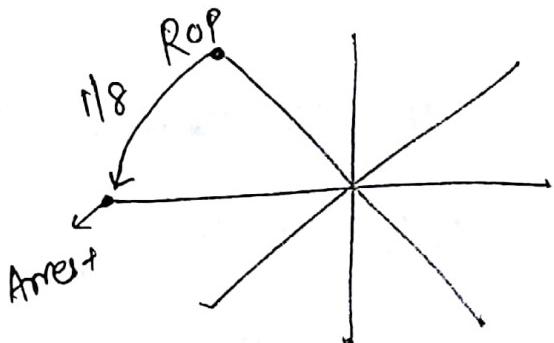


\hookrightarrow so take much time; slow progress of labour
 ↓

Rx: Wait & Watch; When baby is Anterior then delivers

2nd outcome \Rightarrow Deep transverse arrest

\hookrightarrow dt + android Pelvis



↓
do the c.s.

but if Pelvis is N; then
 i) if Inadequate contraction seen

\hookrightarrow give oxytocin

ii) if Contraction is Adequate

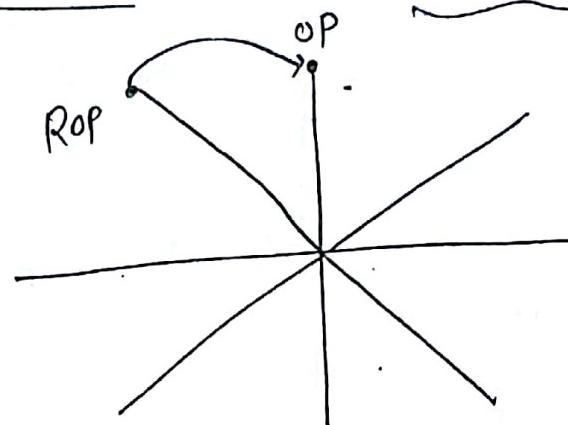
\hookrightarrow Manual Rotation

Forceps Rotation

Vacuum device extraction

3rd outcome

Persistent OP / Direct OP

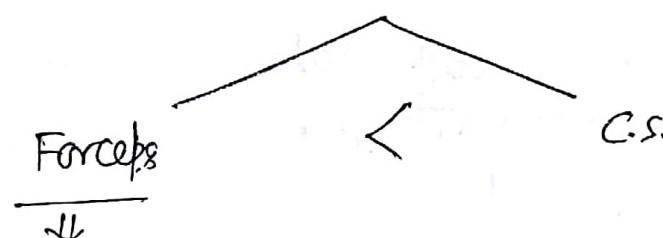


↳ Seen in Anthropoid Pelvis
⑨〇

↓
Mechanism of Labours

↓
"Face to Pelvis" delivery

* In case spontaneous delivery (Not happening)



In Posterior \Rightarrow Incidence of
Position Maternal injuries
& severity rises

* % of babies are in O.P. at onset of = 20%
Labour

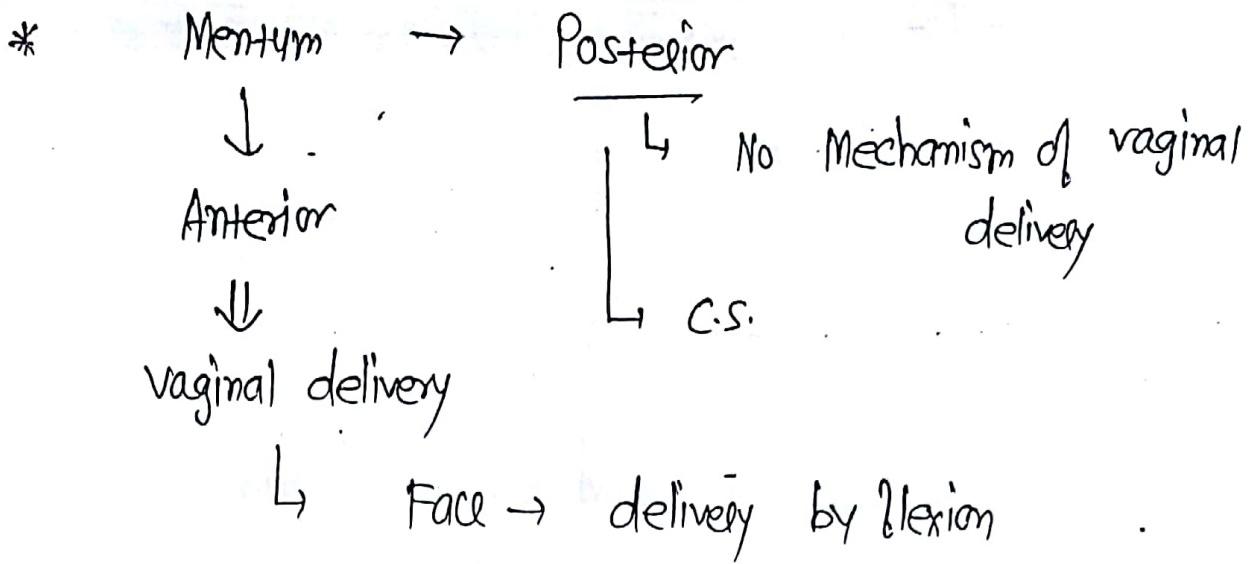
at the end of = 57%
Labour

* FACE / Brow PRESENTATION

Mic cause of Face presentation \Rightarrow Anencephaly;

Pelvis type; which favour it \Rightarrow Platypelloid

Anything that prevents flexion of head \Rightarrow Risk factors (> 1 Loop of cord around
Neck; tight loops /



* Mento posterior in Labour ⇒ C.S.

Mento posterior in early Labour ⇒ wait & watch
↳ to Rotate gives time

* Brow presentation

```

graph TD
    A[Brow presentation] --> B["engaging Diameter ⇒ Mentovertical = 14cm"]
    B --> C[No Mechanism of Labour]
    C --> D[Brow in Labour → C.S.]
    D --> E[Brow in early Labour → wait & watch]
  
```

TRANSVERSE LIE

MIC cause ⇒ PreMaturity

MIC cause at term ⇒ Placenta previa

- Most Commonly Seen in "Platybelloid" Pelvis.
- Highest Risk of cord prolapse

* Transverse Lie — In Labour — do C.S.

↳ Pries ECV

(91)

↳ if Requirement Met.

* Neglected Shoulder

↳ Upper segment \Rightarrow Tonically contracted

Lower Segment \Rightarrow Stretch

Bendis Ring \oplus in b/w us & LUS



case of obstructed Labour

↳ FHR Absent

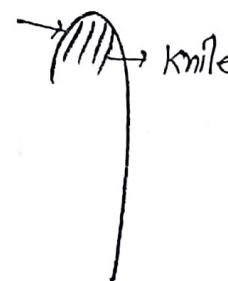
↳ do C.S.

Destructive procedure \Rightarrow i) Evisceration;

ii) Decapitation;



Hook



knife

Q: Lady part in Neglected Shoulder presentation in dead baby.
All the following can be done except \Rightarrow

a) C.S.

b) Evisceration

c) Craniotomy (b/c for if vertex presentation Required)

d) Decapitation her + transverse lie part

Q. Confusion of Breech presentation on P/V examination ↴

Face presentation

↳ frank breech Most commonly confuse.
Gn if ~~the~~^{two} bony prominence & one
opening (Mouth) Makes a triangle;
while In breech it forms straight line

INSTRUMENTAL DELIVERY

- do when we cut short 2nd stage of Labour
- Pre-Requisites ↴

F → Fully dilated Cx

O → No Obstruction in the path

R → Ruptured Membrane

C → Good Uterine Contraction

E → Engaged head / Empty bladder / episiotomy

P → Favourable presentation

■

Forceps delivery

- difficult
- Maternal Exhaustion
- We're in heart disease
- In Fetal distress (Forceps > Vacuum delivery)
- * In general Forceps prefer over vacuum
- In pre-term
 - if P.O. < 34 weeks, vacuum is absolutely C/I
- Face presentation
(MentoAnterior Position)
 - In Face presentation C/I.
- After coming head of breech
(Piper's Forceps)
- More Maternal injuries
- More fetal injuries

Q Which fetal injuries are M/c in Forceps delivery??

- IVH
- Facial Nerve injuries
- Brachial Plexus injuries
- Cornea of the eye

Q Injuries which see in vacuum Not in Forceps delivery??

- 6th Nerve injuries
- Epiphelhematoma
- Retina injuries

Vacuum delivery

→ easier Application (92)

→ Needs Maternal effort

→ also we're in heart disease,
Not C/I

In Forceps delivery)

Forceps prefer over vacuum

→ In pre-term

- if P.O. < 34 weeks, vacuum is absolutely C/I

→ Face presentation
C/I.

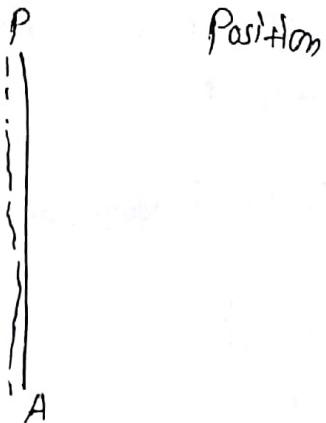
More fetal injuries

* Classification of Forceps delivery \Rightarrow

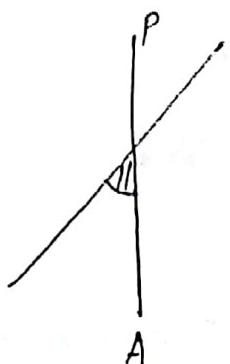
| | | |
|-------------------------|-----------|-----------------|
| Outlet | $\geq +3$ | |
| Low | +2 | |
| Mid Cavity - b/w 0 & +2 | | { Not in Modern |
| High - above 0 | | days |

* Criteria for application of outlet forces \Rightarrow

- ① Scalp visible at introitus \nearrow
- ② head on Pelvic floor $\geq +3$
- ③ skull on Perineum
- ④ Sagittal Suture should be preferably in A-P



⑤



Rotational deflection $< 45^\circ$

\hookrightarrow apply outlet forces

How to know that Forceps is correctly applied \Rightarrow



(93)

Blades have to be equidistant from sagittal suture

Along which fetal diameter we apply Forceps blade

\hookrightarrow Occipito Mental (13.5 cm)

* Left blade of Forceps \Rightarrow Introduced first

\hookrightarrow M/c Position = Left,

* M/c outlet forceps used \Rightarrow Wrigley's outlet forceps
 ↑ Not used in After coming head; b/c of short Nature

\downarrow
 for both outlet & Low position,
 short forceps & have English Lock

* Kelland's forceps \Rightarrow for Rotational defect

\hookrightarrow Long forceps

* Piper's forceps \Rightarrow for after coming head

\hookrightarrow Pelineal curve \oplus (Baby body Rest here)
 very long forceps

VACUUM DEVICES

- Use only plastic cup

↳ Bell shaped

diameter = 5-7 cm

Silastic

Pressure = 0.8 kg/cm^2
generated

Centre of the cup @ Flexion point



3 cm Anterior to Posterior fontanelle
OR

6 cm Posterior to Anterior fontanelle
@ Saggital suture

- The Margin of cup touches the Posterior fontanelle

Failed Vacuum \Rightarrow 3 Pulls - No descent of head
3 Rob offs.

* If one device fails are govt for C.S.

C/I of Instrumental delivery \Rightarrow

- a) Contracted Pelvis
- b) CPD.
- c) HIV \oplus ve Patient
- d) K/o Coagulation defect in babies
- e) osteogenesis imperfecta

ANTEPARTUM HEMORRHAGE (APH)

(q4)

- * Bleeding from or into the genital tract beyond the period of viability
 - ↳ In India ≈ 28 weeks

- * Causes of APH → Abruption

Placenta Previa

↳ Placenta Lying in the Lvs

Premature separation of a Normally Located Placenta (from Underlying decidua)

ABRUPTION



Advanced Maternal Age



Smoking



Multiparity

Previous history upto 15y.
(Relative Risk)

Highest Risk

PLACENTA PREVIA



Previous history
(5+)

Pre-eclampsia ; Thrombophilia High Risk
Trauma Folic acid deficiency
Polyhydramnios Fibroid
PRoM
Long standing oligohydramnios

Previous L.S.C.s.

(More the Number higher Risk)

Early ♀ USG

In Previous G.S.

↓
Migration abt

↳ So; RIF for placenta previa

Placenta May be Low Lying

↳ In Subsequent Scan ⇒ Placenta ⇒ Upper segment.

↓ + differential growth of uterus

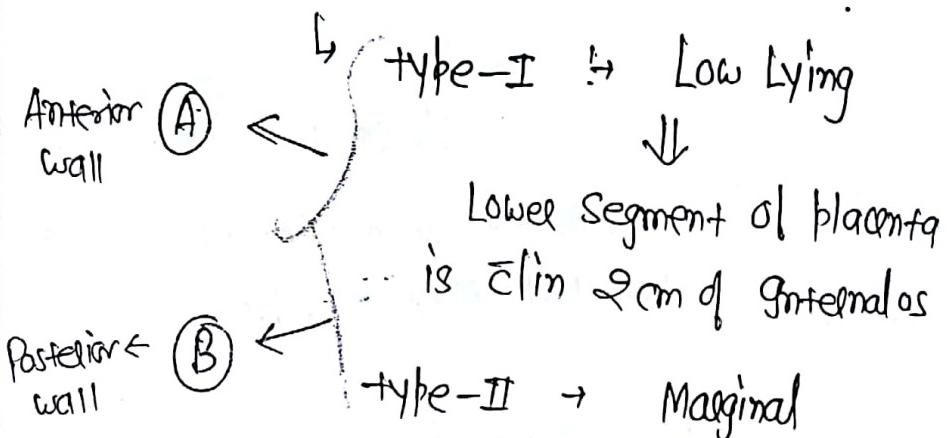
Placenta Migrates

Endometriosis

Mullerian Anomalies

CLASSIFICATION

Placenta PREVIA



Type-III ⇒ Partial (Partially covering the internal os)

Type-IV ⇒ Complete/central (completely covering the internal os)

Other classification ⇒

Minor Placenta previa ⇒ Mode of delivery ⇒ Vaginal / Type 1A; 1B; 2A

Major II ⇒ Type 2B; 3, 4

Mode of delivery ⇒ C/S

* Dangerous Placenta previa ⇒ 2B ⇒ Fetal distress

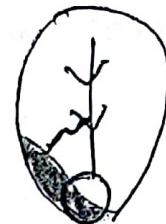
On type 2B ← Startle worthy sign ⇒ Pushing down if head produces a dip in FHR.



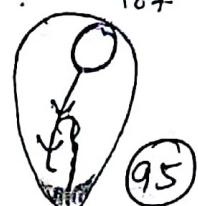
Type I



Type II



Type III

Type IV
95

Classification of Abruptio

Revealed

Blood → extalle out

Concealed

Blood collects behind
placenta (Retroplacental
clot)

Mixed

M/C

* PAGE's classification of Abruptio ⇒

Type 0 ⇒ Retrospective diagnosis,

Type 1 ⇒ bleeding & Pain (FHR ⇒ N)

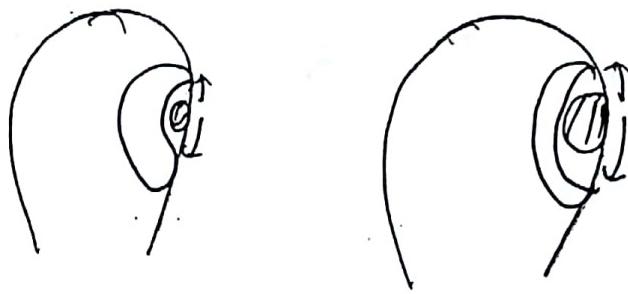
Type 2 ⇒ Bleeding + Pain + Fetal distress

Type 3 ⇒ Bleeding + Pain + GUD + Shock
± DIC

decidua
Retroplacental
clot

Maternal
⇒ tissue thromboplastin
↓
Potent uterotonic
(Pain)

* Abruptio is self propagating



Risk of DIC is dependent on how long Abruptio to delivery times taken.

* Termination of Pregnancy

↳ only Mx of Abruptio



No Role of conservative Mx.

* Pt. comes \in APH; how to tell about P.P. & Abruptio?

P.P.

Bleeding

↳ Painless

Bright Red (Fresh bleeding)

Causeless

Abruptio

Bleeding

↳ Painful

\rightarrow Altered (dark colored)

\rightarrow Preceding event

↳ Pre-eclampsia

Trauma

Hypotension M/c

General Physical examination

\Rightarrow Hypotension is

Not common (bcz basal BP is high)

P.P.
Uterus Relaxed

Non-tense

Non-tender

Fundal height = POG ~~+++~~

< POG (Sometimes)

P/A
examination

Abruption

Uterus tense

tender

(96)

Fundal height > P.O.G.

Less common

Fetal
distress

More Common

Most Common

(Fundal height < POG)

Proneesse Lie

Malpresentation

Less common

H/o warning hemorrhages

No warning hemorrhage
(Acute event)

Should not be done in APH

Until you Rule out placenta
previa

p/s And
pl/v examn

Transabdominal USG

TOC

* TVS is Not CI; but it
is More Sensitive for Posteriorly
Located Placenta

Q8

Pt. comes ∞ APH

\hookrightarrow Pt. Not Loc Lying (Fundal)
No RPC

ans:

Still a Case of Abruptio

\hookrightarrow Plv examination

\hookrightarrow ARM

Confirm



Induce Labour

Blood stained
Liquor

*

Placenta Previa

\hookrightarrow Mx \Rightarrow Are there any indications for TOP
 \Downarrow

Yes (it is in order to do TOP)
 \Downarrow

i) P.P. + Unstable vitals; (Mic indication for TOP)

ii) P.P. + Fetal distress;

iii) P.P. + > 37 weeks.

do it Right Now

iv) P.P. + Continuous Bleeding Per vaginam

v) P.P. + Woman goes into Active Labour

vi) P.P. + GUD

vii) P.P. + GIA (Incompatible for life)

Q. G₃P₂L₂; 34 weeks APH (Bleeding P/v; painless); on examination Fundal height = 34 weeks; Relaxed Non-tender Non-tense; Relaxed uterus; RR = 100/m; BP = 114/76. (97)
USG = type 4 PP.

Mx \Rightarrow Conservative Mx.

- * Indication of conservative APH
 - i) Unstable vitals;
 - ii) Fetal distress;
 - iii) Major degree of Placenta Previa;
 - iv) Far from term (<32 wks)

* conservative Mx in Placenta Previa

MacCallum Regimen

i) Bed Rest

ii) Maternal Monitoring : vitals;

FH (Fundal height)

AC (Abdominal circumference)

BPV (Bleeding Per vagina)

Baseline IX

Uterine contraction

iii) Fetal Monitoring : FHR

iv) if patient <34 weeks \Rightarrow Steroids for Fetal Lung

Dexamethasone (National guideline) \downarrow Maturity \leftarrow

6 mg. i.m. x 4 doses x 12 hr apart.

BetaMethasone - 12 mg x 1m x 2 doses 24 hr apart.

v < 34 wks + Uterine Stimulants (Mild contraction)

↳ give Tocolytics (to buy time for steroids
corel)

↳ also for Ecr

Hyperstimulation

DOC (Tocolytics) \Rightarrow Nifedipine

Safest Tocolytic \Rightarrow "

DOC of Tocolytics in heart disease \Rightarrow Atosiban (oxytocin Receptor Antagonist)

Initially β -Agonists used as Tocolytics

↳ cause \Rightarrow Hyperglycemia AIIMS May 18

Hypokalemia

Tremors

Tachycardia

Amythmia

Cardiac arrest

Pulmonary edema

only β -Agonist used as Tocolytics

Now a days



Terbutaline

Indometacin

↳ Not given > 32 week

Premature closure
of ductus arteriosus

Halothane

Alcohol

Diazoxide

MgSO₄ (acts as Tocolytic @ 9-10 meq.)
↳ given 4-7 meq

it has Neuroprotective action (prevent cerebral palsy)
for preterm babies

toxic dose for Mother

so Not given as a
tocolytic agent.

(98)

End point of conservative Mx

↳ 37 wks Q or any other indication of TOP

ABRUPTION ⇒ No Conservative Mx

↳ Indication for TOP

No Role of Tocolysis

Vaginal delivery is preferred



bc - she has high Risked DIC

Progress very quickly in Labour
(tissue thromboplastin)

Precipitate Labour (entire process of Labour < 3 hr)

do CS; when ⇒ Unstable vitals

Fetal distress

Fal from term

ii) Intraoperatively we see Red Uterus



COUVELAIRE UTERUS

(Bleeding clin Myometrium)

Seen in Uteroplacental Abnormality / Abruptio.



- It is Not an Indication of Hysterectomy
- It is R/F for PPH.

Q. 34 week ♀; Bleeding Plv & Pain; O/E uterus 36 cm; tender; FHR = 100 bpm; on Evaluation BT / CR / INR = deranged
 Mx \Rightarrow It is the case of DIC

Transfuse FFP and try for vaginal delivery.
 In cesarean section; Pt. Bleed profusely from all the sites & dies on table.

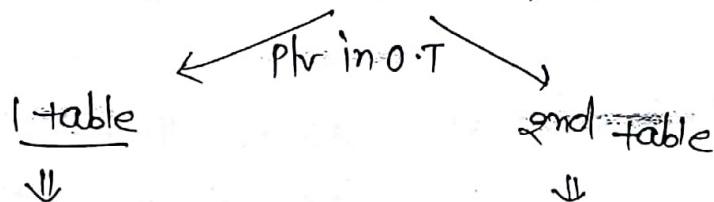
Most cause of DIC in obstetrical \Rightarrow Abruptio.

* Placenta Previa \rightarrow term
 \downarrow

Mx \Rightarrow USG

OR

do "Double set up examination"



Ready for emergency
C.S.

on Plv \Rightarrow Fornices bogginess (≥ 2 fornices)

We are done for Major placenta previa

If we don't get any fornices bogginess



Goes up to the level of internal os



If bleeding \oplus
or placental tissue \ominus
Near Internal os.

If No placental tissue

Near the Internal os
(Minor)

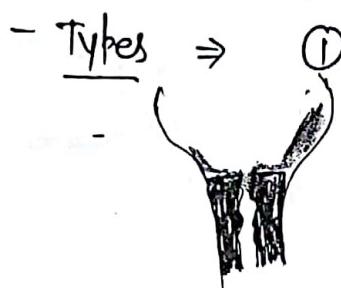


do emergency C.S.

ARM \rightarrow Labour Room

ADHERENT PLACENTA

(N) Placenta is attached to Decidua; but here Placenta is attached to Myometrium & intervening decidua is absent



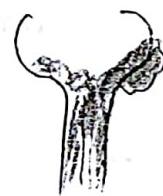
- Types ⇒ ① Accreta ⇒ Attached to Myometrium
↳ Most common

"Large Intraplacental Lacunae" ⇒ Sonological finding

② Increta ⇒ Invades into the Myometrium



③ Perceta ⇒ Penetrates through & through the Myometrium. It comes out in the serosal surface.
↳ Least common



- Highest R/F ⇒ Previous L.S.G.S. + Placenta previa (current)

Q9 Highest R/F for Adherent placenta :

(a) ~~Previous L.S.G.S.~~

(b) Previous P.P.

Other R/F ⇒ Curettage
Scal
Multiparity
Increasing Age

IOC ⇒ USG + Doppler

↳ Heterogenous placenta

↳ Intraplacental Lakes
(Blood tissue in placental tissue)

If we have Any doubt OR we want to know about depth

do MRI

(N) pt. b/w placenta & decidua

on HPE

⇒

Absent / Incompletely developed Niobuchs Membrane
(Layer)

It is fibrinoid degeneration
b/w trophoblast & decidua

Mx ⇒ Keto Adherent Placenta

↳ Elective Cesarean (classical)

+
Hysterectomy

↳ If we incise on Lus,
Myometrium cuts & we
damage placental tissue

• Presentation in Undiagnosed case ⇒
deliver

↳ No sign of Placental separation

↳ If we try to Manual Removal

↓
Pt. bleeds

↳ Refractory PPH (Pt. pt. with Refractory
PPH)

↓
do hysterectomy

* If patient denies hysterectomy

It will go autolytic digestion

c/m 6 months

↳ deliver vaginally

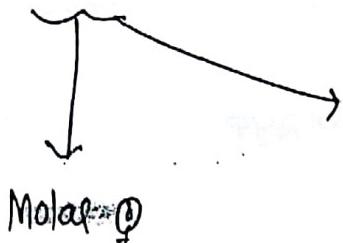
Leave the placenta
on fetus

↳ Cut the cord short as possible as close
to placental end

MOLAR PREGNANCY

(100)

GTD \Rightarrow Gestational Trophoblastic disease



GTD Neoplasia

- Partial
- Complete

- Invasive Mole
- Choriocarcinoma
- PSTT (Placental Site Trophoblastic Tumor)
- ETT (Epithelioid Trophoblastic Tumor)

PARTIAL MOLE

- Chromosomal Make up
of Molar ♀

\hookrightarrow Triplid (90%)
 \hookrightarrow 69 XXY

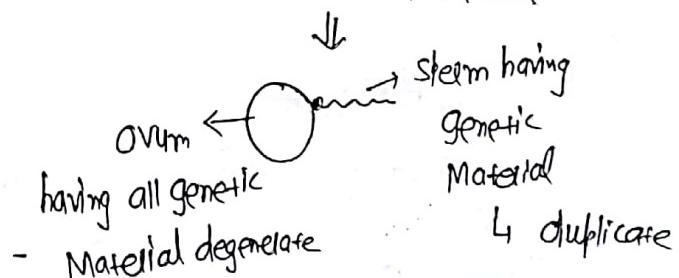
10% Tetraploid

Extra set - Paternal
Dispermic



COMPLETE MOLE

- Diploid ($46XX$) $\xrightarrow{\text{got}}$
- Monospermic ($46XY$) $\xrightarrow{\text{got}}$
- All genetic Material is Paternal



Bleeding

(2nd Trimester ♀)

O/E \Rightarrow Fundal height \leq POG

Micro diagnosis USG

\hookrightarrow Missed Abortion

MC presentation

Bleeding (\approx grades like reside)
(2nd Trimester ♀)

O/E \Rightarrow Fundal height $>$ POG

USG

\hookrightarrow Snow storm appearance

PARTIAL MOLE

Fetus Sem.

↳ dies d/t Multiple Anomalies

* If we can see focal hydroptic in Placenta

↳ Transverse diameter > AP diameter

Molar ♀ Most commonly diagnosed in 1st trimester (USA in 1st trimester)

- HCG > than expected
(but Not Markedly high)

HCG

No Fetus on USG Sem

Ovaries → Theca-lutein

Cyst in it.

↳ 97.25-30% cases ≈

- Markedly Raised (>105)

(HPE)

— Gold Standard

- i) Less Marked trophoblastic Proliferation Investigation (Confirmation of
- ii) Focal hydroptic degeneration diagnosis) of villi
- iii) Fetus +
- iv) Vascular

(HPE)

- i) extensive trophoblastic Proliferation;
- ii) complete hydroptic degeneration of villi
- iii) No Fetus
- iv) Avascular

(-)

- Trophoblastic
Scalloping +

↓

Stromal Inclusion

X

X

X

X

Medical
complication

- i) Thyroid Storm → d/t high hch
- ii) Pulmonary embolism
- iii) Early onset pre-eclampsia
d/t Paternally derived Ag.
- iv) Hyperemesis Gravidarum
↳ d/t hch

- Partial
 Partial Mole \rightarrow GTN (3-5+) Complete Mole \rightarrow GTN (15-20+) 199
101
 Partial Mole \rightarrow Chorio.ca (< 14) Complete Mole \rightarrow Chorio.ca (4-17)

Mx \Rightarrow Suction & evacuation (do till fundal height \leq 12 weeks)

if pt is \geq 40 yrs + complete Mole \oplus

\hookrightarrow do Hysterectomy (risk of chorio-ca)

SLE \Rightarrow Pulmonary Embolism

Thyroid Storm

to prevent Embolism \Rightarrow Start suctioning first then followed by oxytocin.

Check curettage \Rightarrow Sharp

* Send sample for HPE

\downarrow
then follow up w/ HCG

Weekly S: HCG \Rightarrow until 3 (\ominus) values

Total \Rightarrow up to 6 months
Surveillance \downarrow Monthly HCG

* Avg. time to β -hCG to become (\ominus) \Rightarrow 9 weeks
Avg. time to β -hCG to become (\ominus) in Partial Mole \Rightarrow 7 weeks

Say patient Not to conceive

GVD Avoid
bleeding

\hookrightarrow Contraceptive of choice \Rightarrow OCP
Barrier can be used

* Criteria for Diagnosis of GTN (Any 1) ↴

① 4 consecutive HCG values shall shows of Plateau

↓
91 → 114 → 101

(Less than $\pm 10\%$ values)

② 3 consecutive HCG value that shows a Rise

↓
114 → 141 → 101

③ HCG Remains Elevated even after 6 months of Suction & evacuation

④ HPE → GTN

* Clinical presentation of GTN ↴

i> Bleeding Per vagina — Persistent

ii> Shock

iii> Persistent Theca Lutein Cyst

↓

④ Theca Lutein cyst shows Spontaneous Resolution
Clin 2-4 months of Suction & evacuation.

iv Uterine Subinvolution

↳ Uterus doesn't go to Non Q state after
Suction & evacuation

v Metastasis

* High Risk for conversion of GTN \Rightarrow

- i) ≥ 35 yr (Maternal Age)
- ii) Fundal height \rightarrow POG
- iii) HGS ≥ 105
- iv) Bl Large (> 6 cm) theca Lutein cyst

102

Should Receive
Prophylactically
Chemotherapy
 \Downarrow
Actinomycin
OR
Methotrexate

* M/c GTN \Rightarrow Invasive Mole

4c GTN \Rightarrow ETT

M/c GTN to develop after Full term ♀ \Rightarrow Choriocarcinoma

* Choriocarcinoma M/c develops after
which type of ♀ \Rightarrow Complete Mole

Malignant

Metastasis seen (Common ball Metastasis on CXR)

\hookrightarrow M/c site \Rightarrow Lung $>$ vagina $>$ Liver $>$ Brain

2nd M/c finding on Choriocarcinoma
on CXR Lung \Rightarrow "Snow Storm Appearance"

* In Vagina

\hookrightarrow Bluish Submucosal Nodules (+)

\hookrightarrow don't take biopsy b/c very vascular

* How to differentiate Invasive Mole from Choriocarcinoma?

(HPE)

Presence of villi \Rightarrow Invasive Mole

Absent villi \Rightarrow Choriocarcinoma

Choriocarcinoma

- M/c
- M/c after complete mole

Tumor \Rightarrow HCG
Marker

Malignant

Syncytiotrophoblast
(Cytotrophoblast)

- Hemorrhage / Necrosis +
- Chemo sensitive
- TOC \Rightarrow Chemotherapy

PSTT

Rare

M/c after FFP

Full term ♀

Human Placental
Lactogen

HPE \Rightarrow PLAP

Placental alkaline
phosphate

70% Benign

Mononuclear = Monomorphically
(No + Syncytial ones)

ChemoResistant

Hysterectomy

* STAGING OF GTN \Rightarrow

Stage I \Rightarrow Tumor is confined to uterus

Stage II \Rightarrow Outside uterus but clin pelvis

Stage III \Rightarrow Metastasis to lungs (good prognosis) $\xrightarrow{\text{Metastasis to lungs}}$

Stage IV \Rightarrow Metastasis elsewhere except \rightarrow vagina

(HPE)

Modified Cutta Scoring

\hookrightarrow Low Score

(Good Prognosis)

High Score

(Bad Prognosis)

i) Age

≤ 39 yr

≥ 40 yr

ii) HCG ($\frac{\text{U}}{10,000}$) $\leq 10^3$

$\geq 10^3$

iii) Type of Antecedent

Molar

♀

FTP

(Full term Pregnancy)

iv) Duration from

Clinical month

Antecedent pregnancy

Physiological

v) Site of Metastasis

Lungs

Liver, Brain,

vi) No. of Metastasis

< 4

> 8

vii) Tumor size

< 3 cm

> 5 cm

viii) H/o Previous
Chemotherapy

Single Agent

Multi Agent.

if Total Cutto Score ≤ 6 ≥ 7

↳ Low Risk GTN

High Risk GTN



Single agent chemotherapy



Multi Agent chemotherapy

also in
stage I

Methotrexate



if Pt. Resistant

Actinomycin D

E → Etoposide — D₁
 M → Methotrexate — D₂
 A → Actinomycin D — D₃
 C → Cyclophosphamide — D₄
 O → Oncovin (vinorelbine) — D₅

↓ if Resistant

E → D₁M → D₂A → D₃

E → Etoposide

P → cisplatin

Follow up \Rightarrow Weekly check - till 3 N value

End point of chemo. \Rightarrow 1st N value - 3 more cycles of chemotherapy

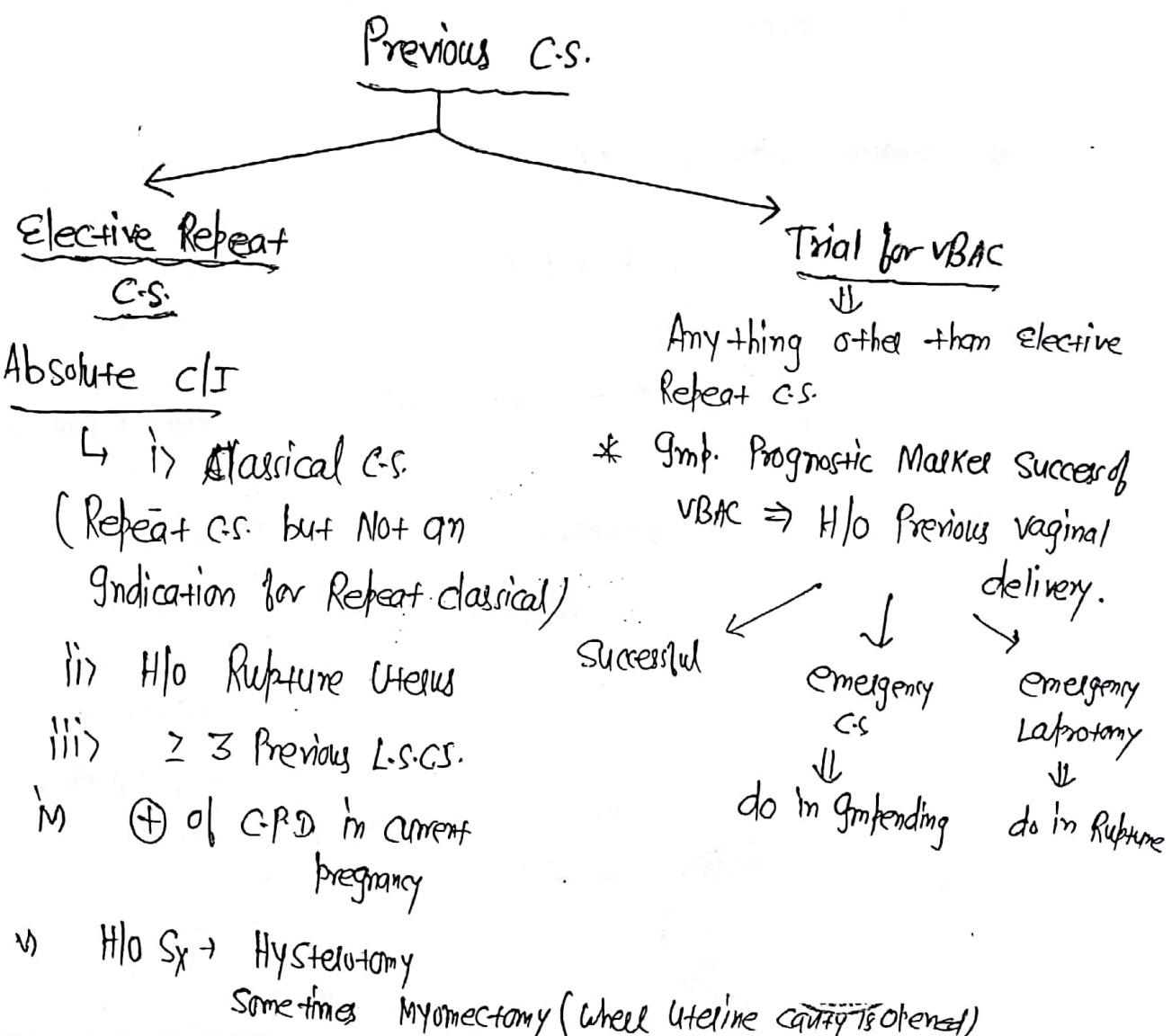
After N value \Rightarrow Monthly follow up

Period of Surveillance for Low Risk GTM - 12 Month

" for high risk " \rightarrow 24 Month

Advice & Contraception of choice is same as Molar Q
↓ ↓
 combined ocp's

Not to conceive



G₁P₁L₁ = previous L.S.C.S.; done for C.P.D. 40wk; Head is bree bloating Cx os closed ^{Unellaxed} v M_x

(104)



C.S. (b/c C.P.D. + in current ♀)

vii Whole vaginal delivery is dI (Contracted Pelvis
Major degree of Placenta previa)

* Relative Indication for Repeat C.S. (C.S. > V.D.) :

- i) Previous L.S.C.S. — ∞ Breech in current ♀
- ii) " " — ∞ Macrosomia
- iii) " " — ∞ Post-term

* Sign & Symptom of Impending Rupture :

i) Non Reassuring FHR

↳ 1st FHR changes \Rightarrow Tachycardia

M/c FHR changes \Rightarrow Bradycaida

- ii) evidence of Maternal Tachycardia;
- iii) evidence of Scal tenderness (Pain);
- become only significant if Non-Reassuring FHR + .



ii) Intraoperatively ; at the Scal site — all Layer of Myometrium have given away but overlying serosa intact

Very Grt. br Next ♀

← "Scal Dehiscence"

* Sign & Symptoms of Rupture of Uterus →

- i) Maternal Tachycardia;
- ii) ↓ Hypotension;
- iii) Severe fetal distress / FHR absent;
- iv) Fetal parts all superficially felt, (as the fetus is expelled into Abdominal cavity)
- v) Uterine contour is lost.
- vi) Sudden stoppage of uterine contraction;
- vii) fresh bleeding per vagina;
- viii) Catheterise → gross hematuria
- ix) Loss of station (Most characteristic feature of Rupture uterus)

\Rightarrow Emergency Laparotomy (Tries to Repair)

* Can we do induction in Labour (IOL) in previous CS

↳ Not C/I

↓

Spontaneous Labour > IOL

↓

DOC \Rightarrow Oxytocin

Not be given \Rightarrow Misoprostol

* Can we do augmentation of Labour

↳ Not C/I

↳ Continuous fetal Monitoring (+ to identify 1st sign of impending Rupture)

* Can we do ECV in Previous Cs.

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↳ Yes (Not C/I)

(105)

Relative C/I for ECV

* Internal Podalic version (IPV) in Previous Cs

↳ Absolutely C/I in Previous Cs.

* Induction of Labour is C/I in → Contracted Pelvis

↓
It Means we can't deliver vaginally.

Classical Cs.

H/o Rupture uterus;

Transverse lie;

Major degree of Placenta previa;

Category 3 FHR tracing

↳ ominous to baby

↳ Immediate delivery

* Pre-Induction Score → to induce labour

(Bishop's Score)

Score

| <u>Cervix</u> | 0 | 1 | 2 | 3 |
|-------------------------|-----------|--------------|----------|--------|
| Position (Leut. Imp.) | Posterior | Mid-position | Anterior | |
| Consistency | Firm | Medium | Soft | |
| Effacement | 0-30% | 30-50% | 60-70% | > 80% |
| Dilatation (Most. Imp.) | Closed | 1-2cm | 3-4cm | > 5cm |
| Baby's station | -3 | -2 | -1, 0 | +1, +2 |

In Modified Bishop \Rightarrow Cx effacement is Replaced by Cx Length

In Simplified Bishop \Rightarrow dilatation
effacement
station

* All Scores $\geq 9 (> 8) \Rightarrow$ Favourable \rightarrow Initiate Uterine contraction.
 $\leq 5 (< 6) \Rightarrow$ Unfavourable

Total Score - 13

Initiate Uterine contraction

1. firstly do cervical Rikening

Medical Methods

Mechanical Methods

- Cerviprome gel
(Dinoprostone)
PGE₂

0.5mg

Ideal \rightarrow Intracervical
Acceptable \rightarrow Posterior fornix

- Put 6 hrly Maxm 3 doses
In 24 hrs

- Mlc used
- If do only Rikening; so, after Rikening; give oxytocin + to Uterine contraction (after 6 hr.)

1. Better
(do Rikening + Uterine contraction)
- after 4 hrs we give oxytocin; if Uterine contraction is Not

- Laminaria Tents
(osmotic dilators)

- Bulb of Foley catheters

• Misoprostol
(PGE₁)

25 µg/1
vaginally

- Put every 4 hrly
Maxm 6 doses/24 hrs.

TWINS PREGNANCY

209

106

- * M/c type \Rightarrow Dizygotic (70%); k/a "Fraternal twins"
Monozygotic (30%); k/a "Identical twins"

- * In ART procedure \Rightarrow M/c type \Rightarrow Dizygotic twins

- * Incidence of Dizygotic twins

Varies

depend on \Rightarrow Race

ethnicity

Family History

ART

Incidence of Monozygotic twins

Constant

M/c type of Monozygotic twins

\hookrightarrow MCDA

- * Dizygotic \Rightarrow Dichorionic

Diamniotic

M/c type \uparrow

- depend on time of cell division

Clin 7-8 hrs of Fertilization \Rightarrow DC DA

Clin 4th-8th day " \Rightarrow MCDA

Clin 8-12th day " \Rightarrow MCMA

Clin >12th day " \Rightarrow Conjoined twin (Siamese)

- * Monochorionic twins has higher Risk as compared to Dichorionic

- * Marker of Dichorionicity (to know chorionicity we do USG in 1st trimester ($>7wk$))

\hookrightarrow i) 2 separate Placenta on USG

ii) Opposite Sex twins;

iii) Twin Peak sign (Lambda sign)
(Seen 10-14 week)

\hookrightarrow b/c chorion enters into

* In Monochorionic twins

↳ Inverted "T" sign seen on USG

IV) 4 Layers in dividing Membrane

2 Amnion 2 chorion

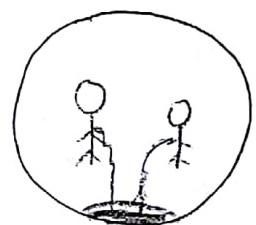
V) Thickness of dividing Membrane



≥ 2mm thick ⇒ Dihorionic

If < 2mm thick ⇒ Monochorionic

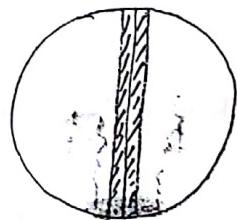
* Monochorionic Monoamniotic twins



* M/c type of Conjoined twins ⇒ Thoracopagus

↳ to join

* Monochorionic Diamniotic twins



* M/c Risk to fetus in twin ♀ ↴

also ⇒

Preterm Labour ↴

GICA ↑

GUGR

} combination to
singleton ♀

* 2 things not seen in Multifetal ♀

↳ Post dated ♂
Macrosomia

* Fetal Reduction ⇒

- ideal time to do ⇒ 10-13 week
- Under USG guided inject KCl into thorax
- converted into 2 fetus (Not less than twins)

* Monochorionic twin (specific) complication ⇒

- TTTS
- TRAP (Twin Reversed Arterial Perfusion)
 - ↳ k/a "Acardiac twinning"
- TAPS (twin Anemia Polycythemia sequence)
- Selective GUGR
- ↑ Risk of GCA

TTTS (Twin-twin Transfusion Syndrome)

- It is seen in Monochorionic Diamniotic

↳ Reason for TTTS

↳ Vascular Anastomosis

Deep artery of one baby anastomoses
with deep vein of other baby

* In Monochorionic Monoamniotic twins

↳ Plenty of Superficial Artery-Artery

Anastomosis (+); so; TTTS

not seen in it.

Diagnosis \Rightarrow USG

Simultaneously \rightarrow Polyhydramnios (by QVP Method
we see Amniotic fluid)
olohydramnios

Deep Artery \leftrightarrow Deep vein

Donor

\downarrow
oliguria
 \downarrow oligohydramnios
Heart failure

Recipient

\downarrow + blood volume
polyuria
 \downarrow Polyhydramnios
CHF
 \downarrow d/+ cardiac overload

* SUINTERO STAGING \Rightarrow Staging of TTTS

Stage 1 \rightarrow Poly/oligo + Bladder of oligo twin is visible + Doppler (N)

Stage 2 \rightarrow Poly/oligo + Bladder of oligo twin is Not visible + Doppler (N)

Stage 3 \rightarrow Poly/oligo + bladder Not visible + Doppler (AbN)

Stage 4 \rightarrow Either / both hydrops fetalis

Stage 5 \rightarrow Either / both gus.

TT \Rightarrow FetoSonic Laser Ablation of the vascular Anastomosis.

(108)

Stuck twin \rightarrow alive; only stuck

Oligo twin of twin-twin transfusion Sx.
can't move; so looks like stuck

Vanishing Twin \rightarrow Spontaneous abortion of one of the twin.

Fetus Papyraceus \rightarrow One twin dies & is compressed by other twin.

Grimacing death

\downarrow
do TOP

\downarrow
take out both the babies; as other baby may go to hypoxic injury

if one twin - already die

\downarrow
L gd

continues pregnancy

+ monitors the other twin

\downarrow
Baby may go hypoxic injury & coagulation defect both

Superfetation \rightarrow different cycle (Menstrual)

Superfecundation \rightarrow same cycle

OUTCOME OF TWINS \rightarrow Lie of 1st Twin

1st - Twin - Longitudinal - vaginal delivery

Non-Longitudinal - C.S.

* M/c combination in Labour \Rightarrow Vetter - Vetter
1st twin 2nd twin

Vertex - breech

Q&A $M_x = ??$ if 1st twin = breech; 2nd = vertex

Vaginal delivery is Not c/I

C.S. > vaginal delivery

Complication \Rightarrow twin interlocking

Q&A $M_x = ??$ if 1st vertex delivered; 2nd breech



Assisted breech vaginal delivery

Q&A $M_x = ??$ if 1st vertex delivered; 2nd transverse lie



do Internal Podalic Version (IPV)

Breech extraction

↳ i.e. No efforts by Mother at all

gives under
G.A (General Anesthesia);

Mother have no effect
to push the baby; so; do
Breech extraction

* The lie of 2nd baby is Not
decided until 1st baby is delivered

Q&A M/c complication of Monoamniotic twins are

↳ Cord Entanglement

↳ delivered by C.S.

↳ by 32-34 weeks

(109)

Rh -ve PREGNANCY

Husband +ve

both Require
for complication

occur in 1st ♀ @
delivery

Fetomaternal hemorrhage (FMH)

Sensitization of the Mother

0.1ml blood is Required to sensitization

⇒ 1st Ab produced by Mother after sensitization

IgM (doesn't cross the
placenta & Not cause
effect in baby in 1st ♀)

Later she produce IgG (cross placenta)

⇒

Significant fitne Require to damage fetus



GCT done
+ve -ve

Sensitized Non-sensitized

Significant Anemia in fetus

* Anti-D prevents
↓
Sensitization.

dose: $\frac{\text{FMH} < 12 \text{ wks}}{50 \text{ Mgm}}$

$\frac{\text{FMH} > 12 \text{ wks}}{300 \text{ Mgm}}$

MCA doppler tells about it

Peak systolic velocity

$\frac{\text{PSV} \geq 1.5 \text{ Mpm}}{\text{Multiple of Median}}$

cause heart failure

Hydrops

death of fetus

* 300 Mgm of Anti-D Neutralize

| |
|--------------------|
| fetal blood - 30mL |
| fetal RBC - 15mL |

* if we suspect FMH more than usual then to calculate dose of Anti-D — Kb test
(Kleihauer-Batke)

Case I

Rh -ve



Husband +ve



@ 12 week GCT -ve

(Not sensitized)



GCT (28wk)

L -ve



Anti-D 300-Mgm → Prophylactically

↳ this works for 12wks ; so; she delivers after safety.



if Baby blood group +ve



Anti-D 300 Mgm (ideally clin 72hrs ; can be given up to 28 days) i/m.

(110)

Case 2 ↴

Rh One



Husband Rh One

@ 12 weeks

GCT +ve
($< 1:16$)

(Anti-D has No Role in GCT +ve Patient; it Means Sensitization already takes place)



Repeat GCT x 4 weekly

if Rising trend; Repeat 2 weekly



deliver at term

Case 3 ↴

Rh One



Husband Rh One



if Hb level < 5 gm%
of fetus



Hydrops fetalis

@ 28 weeks GCT +ve
($> 1:16$)

↓ followed up for severe Anemia

PSV - MCA Doppler

≥ 1.5 Mon

P.O.G. ≥ 34 weeks

↓
deliver

< 1.5 Mon

↓ Repeat MCA Doppler

P.O.G. < 34 weeks
↓ do cordocentesis
Intrauterine
transfusion ↓ Hb < 8 gm%

Hydrops fetalis \Rightarrow Most common cause (Non-immune Mediated)
↳ CVS - abnormalities
↳ M/c/c
↳ Immune Mediated

Infection that can cause hydrops fetalis \Rightarrow Parvovirus B-19

USG diagnosis \Rightarrow Any ≥ 2 of the following

- i) Pleural effusion
 - ii) Pericardial effusion
 - iii) Ascites
 - iv) Subcutaneous edema
- } Criteria

Scalp edema \Rightarrow Buddha sign.

Findings in hydrops fetalis \Rightarrow Placenta Megaly
Polyhydramnios

* Gene for Rh factor Located on \Rightarrow Short Arm of Chr. 1

HEART DISEASE IN PREGNANCY

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(11)

Symptoms

- Orthopnea
- Paroxysmal Nocturnal dyspnea
- exertional dyspnea (Progressive)
↓

It is Physiological; but progressive
exertional dyspnea is Pathological

Signs -

- Cyanosis/ clubbing
- Diastolic Murmur
- Systolic Murmu. > grade 2
- Cardiomegaly
- Atrial fibrillation / Abn Rhythm
- CHF
- Persistently distended Neck veins
- Pulmonary Artery HTN
↳ Loud P_2
- wide split S_2

* M/c heart disease in ♀ \Rightarrow Mitral Stenosis

M/c congenital heart ds in ♀ \Rightarrow ASD

M/c congenital valvular heart disease \Rightarrow Mitral valve Prolapse

* High Mortality Rate (> 50%) in ♀ \rightarrow

- i) Malform Sx \subset Aortic Root Involvement
- ii) Coarctation of Aorta \subset Aortic valve Involvement
- iii) Eisenmenger Syndrome

TOP (Abortion)

IV Severe MS ($< 1.5 \text{ cm}^2$) \rightarrow valve Area

v) NYHA grade 3/4

vi) Ejection fraction < 45%.

* good outcome

- ↳ ASD Corrected TOF
- VSD Mitral valve prolapse
- PDA Ebstein Anomaly

* if a patient has severe MS

- ↳ Ideally goes to - Valve Replacement
 - ↳ Sx preconceptionally
 - ↳ Not be done during ♀
- ↳ if she wants to continue ♀
 - ↳ do Balloon valvotomy
 - ↳ Ideal time
 - ↳ 18-20 weeks

* if she has valve Replacement

↳ Prosthetic heart valve → Pt. is on Anti-coagulation

↓ if she wants to conceive

- ↳ Antidote for heparin
 - ↳ Protamine sulphate
 - ↳ Warfarin has No Antidote
- ↳ 1-12 weeks → Heparin
- ↳ 12-36 weeks → Warfarin (More Potent)
 - ↳ Causes Embryopathy in 1st trimester
 - ↳ Chondrodysplasia punctata
 - ↳ Stippled femoral epiphysis;
 - ↳ If Warfarin given; PPH happen also
- ↳ 36- onset → Heparin

* During Labour → Stop Anticoagulant

(112)

However Restart the Anticoagulant In vaginal delivery ⇒ After
In C.S. ⇒ After 6hr
After 24hr

Restart = Heparin + Warfarin

With draw Heparin; core
-d INR; As; warfarin has
delayed onset of Action

* GENERAL PRINCIPLES ↴

i) Max^m Rise of CHF ↴

Immediate Post partum > 2nd stage > 32 weeks

ii) Vaginal delivery is preferred;

C.S. is Reserved for obstetric indication

iii) Heart disease indication of C.S. ⇒

- i) Marfan's Sx = Aortic Root Enlargement
- ii) Coarctation of aorta = Aortic valve involvement
- iii) Aortic dissection
- iv) Severe AS

In all Ejection fraction is affected

* In Eisenmenger's Sx ⇒ Vaginal delivery tried

* Induction of Labour (gOL) is not contraindicated
in heart disease
↳ gt is safe

however; Spontaneous Labour is over gOL

Q9 38 weeks - Heart failure; risk of Heart disease;

↓
Mx ⇒ stabilize the patient

iv Prophylactically | Left Lateral Posture | O2 by Mask - Readily available

v Restrict iv fluids @ 75 mL/hr

A.S. ⇒ wet side

vi Restrict the plv exams

vii ARM — can be done

Memb. Rubertone — Prophylactic Antibiotics
(AHA guidelines)
↓
Ampicillin + Gentamycin

Vaginal delivery safe

viii Pain Management ⇒ Epidural Analgesia
(Neuraxial)

ix) Cut short the 2nd Stage of Labour



(113)

Forceps delivery > Vacuum delivery

x) Immediately after delivery → Given Grf. Lasix
(to ↓ Preload)
→ Avoid Methergin

Hang down the patient leg from delivery table (to ↑ venous return)

xi) C.S. ⇒ Anesthesia → SLE ⇒ Hypotension
 Severe AR
 Severe AS
 Cytotoxic H.R.
 * In Aortic dissection (emergency condn) do emergency c.s. under G.A.

↳ Epidural Anesthesia; but if Ejection fraction is low; give General Anesthesia

PERIPARTUM CARDIOMYOPATHY

- Development of heart failure Around Labour in a woman ≈ No underlying Heart Disease
- Ejection fraction - Low
- Left ventricle May or Maynt be dilated
- Mainly in Pre-eclampsia patient; also in Multi-fetal ♀; Advanced Maternal age
- Prolactin has some role to develop
- My = Same as Heart failure

Fetal Monitoring

Fetal Movements \rightarrow Quickening (1st Fetal Movement)

↳ 16 weeks - Primigravida

18 weeks - Multigravida

(N) ≥ 10 fetal Movement in a 2 hr Period of Rest

OR

≥ 10 Fetal Movement in a 12 hr Period in Routine activity

Max^m fetal Movement \Rightarrow @ 32 weeks
Perceived by women

- earliest time for gud in Absence Fetal Movement = 12 hrs
Max^m time for gud in Absence Fetal Movement = 48 hrs

- Modified Biophysical Profile (BPP) \rightarrow No Fetal Movement beyond 32 weeks

also known "cardio-tocography"

NST

>>

AF

\Rightarrow Amniotic fluid

In Acute Injury

In Chronic Injury (UPI)

(N) Heart Rate = 110-160 beats/min.

Beat to beat variability = 5-25 beats/min

Acceleration \Rightarrow ↑ Heart Rate by 15 beats/min above baseline for 15 sec

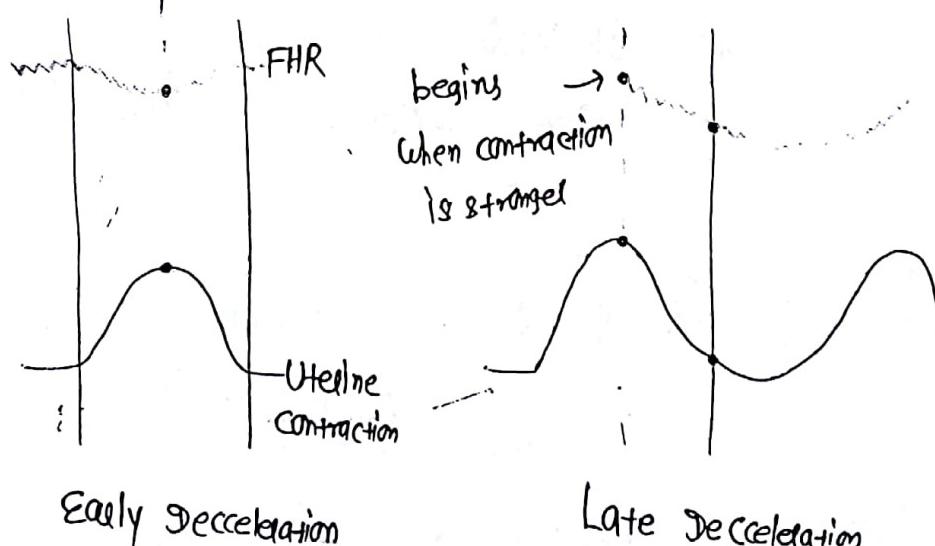
Deceleration

(3 types)

gradual
dip in heart
Rateit is
also bad
outcomeEarly \Rightarrow dip in heart Rate (d/t head compression,Late \Rightarrow dip persisting after the uterine (d/t uteroplacental
contraction ends) insufficiencyVariable \Rightarrow Abrupt dip in heart Rate (d/t cord
compression)Sinusoidal \Rightarrow Marker of Severe fetal Anemia
Severe fetal hypoxia↳ Most ominous type of Pattern
(worst outcome)

When variable deceleration is significant

- ↳ i) if $HR < 70 \text{ beats/min} \times 1 \text{ min}$
- ii) Persistent Around $\geq 50\%$ of uterine contraction



Early Deceleration

Late Deceleration

Reactive NST \Rightarrow two or more than two accelerations
in a span of 20 min.

Non-Reactive NST \Rightarrow < 2 acceleration in a span of
 40 mins
Physiologically if baby is sleeping

* Category 1

beat-
Beat +
variability
 \circlearrowleft N

Category 2



Absent C

Immediate delivery \rightarrow CS. \nwarrow ominous

Category 3

Any one of the following

\downarrow
Brady cardia

Late deceleration

Persistent variable deceleration

Sinusoidal

Further assessment

LLP (Left Lateral Posture)

O₂ by Mask

Stop oxytocin

IV fluids

Give tocolytics

NST in high Risk ♀ \Rightarrow twice a week
(once in 72 hrs)

* BPP (Biophysical Profile) \Rightarrow Manning Score

\nwarrow Report card of fetus

(15)

→ USG for 30 min



We a score of +2 or 3/10

- It has 5 components



Breathing Movement \Rightarrow At least one Movement Lasting 30 sec

+2

Gross Body Movement \Rightarrow 3 Movements

+2

Tone \Rightarrow Flexion - Extension - Flexion

+2

Amniotic fluid \Rightarrow At least 1 Pocket of 2 cm

+2

NST \Rightarrow Reactive

+2

If Score is $\frac{8,10}{10} \Rightarrow \textcircled{N}$

$\frac{6}{10} \Rightarrow \textcircled{N}$ Liquor = equivocal = Repeat testing
On the same day

$\leq \frac{4}{10} =$ Immediate delivery

* \textcircled{AbN} In Acute hypoxic



Loss of Acceleration \rightarrow Breathing Movement \rightarrow Gross body movement \rightarrow tone
1st to become \textcircled{AbN}

* How frequently to be done in high Risk ♀

↳ once in week

DOPPLER

⇒ M/C 1st vessel ⇒ Uptake A
↳ signs of UPI

• S/D Ratio

↳ (N) ♀ S/D ↓ing

• UPI ⇒ S/D ↑es

↓

> 28-30 wk

↓

≥ 3

UPI

R EDF → TOP

• AEDF - TOP ≥ 34 weeks

↳ @ 34 weeks - gives steroid + further monitoring.
or less than 34 weeks

* MCA Doppler ⇒ Not best for UPI

↳ (N); blc in UPI ; foetus sends blood to vitals organs
↓

Early stage MCA doppler ⇒ (N)

↓
Brain sparing effect

* REDF — Umbilical Artery

↳ Steroids - 2 days

Last vessel to shows REDF

Reversal in Venous Doppler



Indicates impending death.

* MCA doppler is best studied for fetal Anemia

* Best for Fetal Monitoring

↳ Fetal Scalp blood pH

(N) \Rightarrow 7.25 - 7.35

Repetitive after 30 min

7.25 - 7.20 = Borderline

$< 7.20 \Rightarrow$ Acidosis

↳ Immediate delivery

* VAST (vibroacoustic Stimulation test) \Rightarrow

High Intensity Sound Waves

Released from Artificial larynx \rightarrow on Maternal Abdomen
for 1 sec (to 2 sec)

(N) Response \Rightarrow Res from baseline by 15 bpm \pm in 15 sec
of stimulus

* MIC Method for Intrapartum Monitoring ↗

| Intermittent Heart Rate Auscultation. | | |
|---------------------------------------|------------------|------------------|
| | <u>1st Stage</u> | <u>2nd Stage</u> |
| In Low Risk ♀ | - every 30min | every 15min |
| In High Risk ♀ | - every 15min | - every 5min |

* Time to Heart Rate listen → Immediately after contraction
& Listen for 1 minutes
(Not 15 sec x 4)

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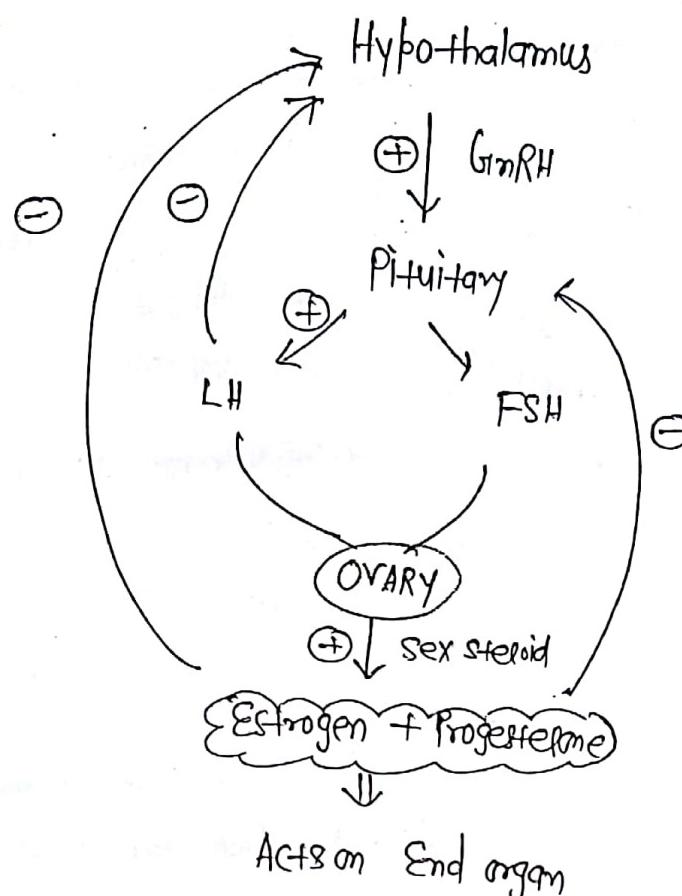
Gynaecology → Base of Gynaec

- 233

↳ Hormones

(118)

* Hypothalamic-Pituitary-Ovary Axis (HPO axis) :



Not develop before Puberty.
Sensitive Around = 8-12 yr
Fully established by = 13-14 yr

Feed forward
Loops

* In absence of / Pubertal change occurs early, ↓Leptin

↳ Hormone Made by Adipose cells that inhibits hunger.

* Estrogen

* C₁₈ Steroids

Types ⇒ E₁ - Estrone - Predominant Estrogen
 E₂ - Estradiol → in Post Menopausal
 E₃ - Estriol → in Reproductive Life
 ↴ in Pregnancy

Most Potent ⇒ E₂ > E₁ > E₃.
 (Natural)

* Most Potent (synthetic)

↳ Ethynodiol-Drostanolone (EDD)

↳ Used in Combined OC's

Progesterone

C₂₁ Steroids

Progesterone
Natural

Progesterone

Synthetic

C₁₉ Steroids
(Androgen derived)

OC's contain it

* Classification of OCP's on the basis of Amount of Estrogen

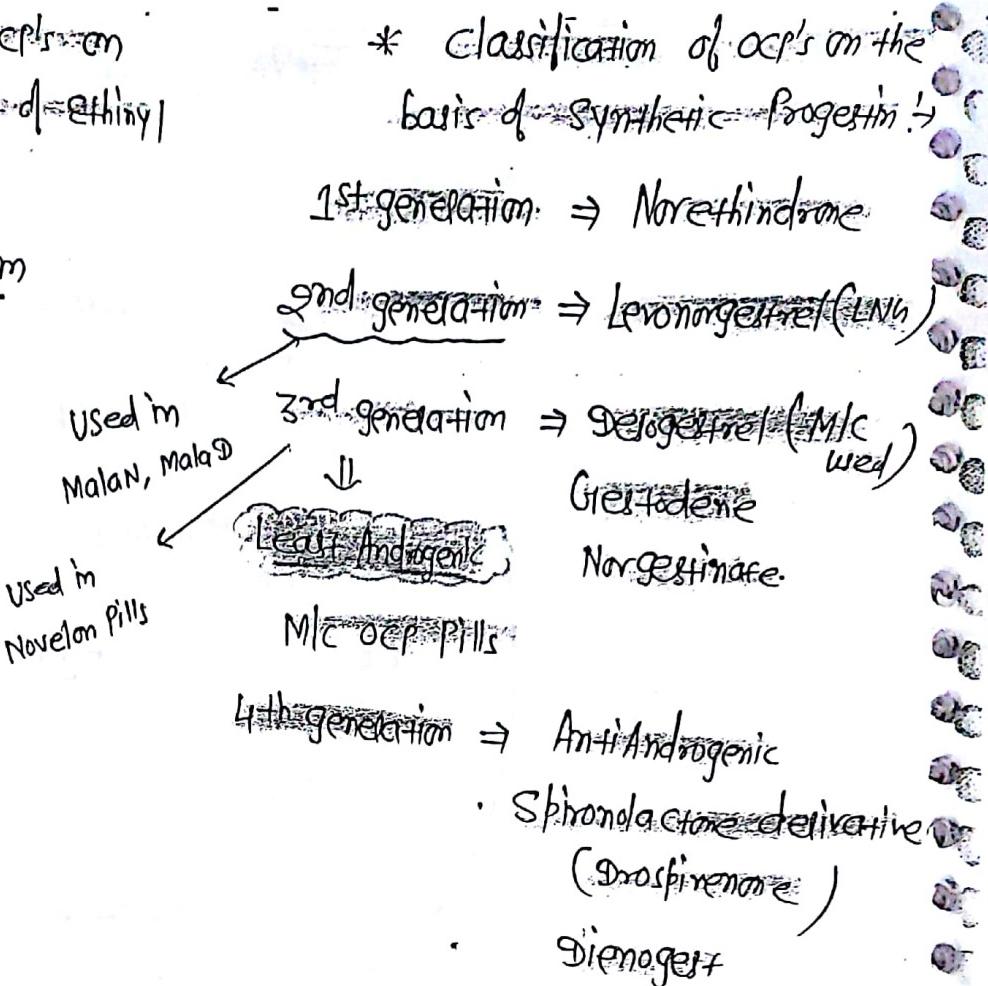
Estradiol \Rightarrow

High dose $\geq 50 \text{ Mgm}$

Low dose $80-35 \text{ Mgm}$

Very low dose $\leq 20 \text{ Mgm}$

Lowest dose 10 Mgm
(LoLoestrin)



as generation goes, Libidinol-like side effect
less & Androgenic side effect also less

* Sources

$E_1 \Rightarrow$ Post Menopausal

\downarrow
Peripheral conversion

Androstenedione $\xrightarrow{\text{Adipose, E}_1}$
 $\xrightarrow{\text{tissue}}$
(Aromatase)

$E_2 \Rightarrow$ Reproductive Age

\downarrow
• Comes from granulosa cells
of ovary \downarrow dependent on
Theca cells

• Corpus luteum

* Sources

• Ovary

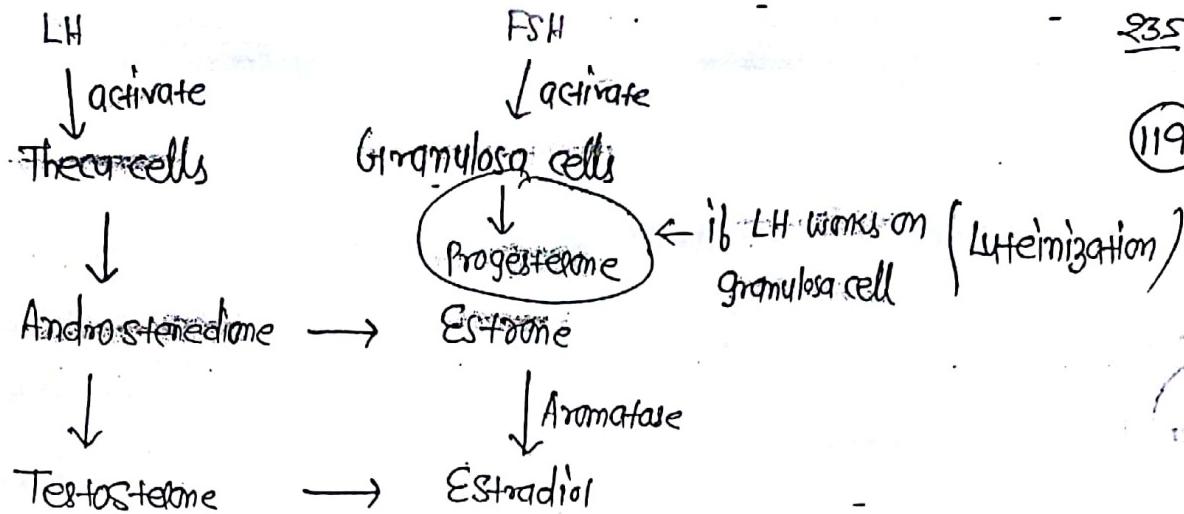
\downarrow Corpus luteum

\downarrow
Formed by Luteinization of
Granulosa cells.

• In $\varnothing \Rightarrow$ Produced by placenta

\downarrow
Precursor (Maternal LDL
Cholesterol)

2 cell 2 gonadotrophin
Theory.



* Theca cell don't have Aromatase enzyme.

* Granulosa cells don't have 17-OHase enzyme; so can't make Androgen

* Placenta - E_3 (Fetal SHBG)
(Pregnancy) E_2

* Most of estrogen → is in bound form (99%)

If free form

Mainly bound =

SHBG

* Bound; ~~or~~ free
↳ Mainly Albumin

(cortisol binding ← CBG
globulin) Not with SHBG

End product

↳ Pregnenediol

also: \leftarrow Albumin
Estrogen $\xrightarrow{\text{SHBG}}$ Sex hormone binding
 \rightarrow (Liver) Globulin
synthesis

End products \Rightarrow Glucuronides
(Sulphonides)

* Receptor II Location

↳ Intracellular

Intracellular

Uterus

Non-Pregnant

(E)

Endometrial Proliferation

(P)

Protective

Stops proliferation

↳ do secretory changes

↓ deciduallisation

Growth of uterus

Growth of uterus

↓

Relaxation of uterine

Smooth Muscle

Cervical
Mucus

Thin

Copious (Large Amount)

Watery

No Spinn barkeit

Thick

Scanty

highly viscous

Elastic can be stretched
b/w fingers

Spinn barkeit ()

↑
(X) Tack phenomenon

high amounts of
NaCl Required
estrogen to see if

Ferning → Fern like pattern
earliest @ 24 hrs
Disappears by 36 hrs
Krause "Arborization".

(X) (No ferning)

Cx Mucus breaks
on stretching

* Peliovulatory Cx Mucus is (E) type \Rightarrow highly brittle.
↳ test of ovulation

* Mechanism of POP's (Minibills)

↳ to make the Cx Mucus thick
(Alteration of Cx Mucus)

* Cx Mucus \Rightarrow Natural Method of contraception
↳ "Billing's Method of contraception"

Q8 Which Natural Method is best (least failure rate)

(120)

↓

Sympathetic

↓

Cx Mucus Basal body temp.
 (Progesterone rises by 0.5°F)

- Cx Mucus is also impermeable to micro-organism
 (P) type ↓
 Natural defense mechanism
- Mirena will less the Risk of Pelvic inflammatory disease

(E)

Fallopian
Tube

↑ Motility of
Fallopian tube

↓ Secretion

(P)

↑ Semen

↓ Motility

Pop → RIF for ectopic

Vaginal Cytology \Rightarrow

Big cell Nuclei become small;
 Subepithelial cells Pyknotic & shades
 ↓
 In Post-Menopausal \Rightarrow Parabasal/Basal cells
 ↓ Small cell;
 Big Nucleus.

Chlorplasm may contain the vacuoles
 High karyokinetic index (Small Nucleus)

Tells about Hormonal Status
 ↑ Parabasal cell : Intermediate cell : superficial cell

Maturation Index

0% : 90% - 100% \Rightarrow ♀

0% : 30% : 70% \Rightarrow Periovulatory ♀

100% : 0% : 0% \Rightarrow Post Menopausal
Post partum,

(E)

(P)

effect on
salt & water

Retention

Excretion

cholesterol

\uparrow HDL

\downarrow HDL

\downarrow LDL

\uparrow LDL

\uparrow Triglyceride

(Total cholesterol yes)
 \downarrow

Cardio protective

Bones

Causes mineralization
of bone

No effect on bone

- Epiphyseal closure

[Post Menopausal \rightarrow Osteoporosis]
 [Precocious puberty \rightarrow ↓ growth]

S. cat² Level

\downarrow

Urinary excretion

\downarrow

Coagulation
Profile

(E) Hypercoagulable State Inhibits Fibrinolysis
2, 4, 8, 10

- No effect,
- so, can be used

H/o Venous Thromboembolism

Stroke
CAD

all absolute
C/I of OCP.

(121)

(E) Estrogen → Causes Upregulation of Progesterone Receptors in the Endometrium

(P) Progestrone → Down Regulation of Estrogen Receptors in the Endometrium

Progestrone acts only on Estrogen primed Endometrium

Estrogen affects to higher centre ⇒

(E) +ve → FSH

(E) In Low Amount +ve → LH

Neuroendocrine phenomenon

In High Amount, LH

+ve

Initiation

LH Surge

High Amount
of Estrogen

(P) Low Amount +ve → LH/FSH

High Amount -ve → LH/FSH



GONADOTROPINS

- Released by Anterior pituitary;
- Basophils cells secrete
- Pulsatile
- Protein hormone



FSH $\tau_{1/2}$ = 3-4 hr

LH $\tau_{1/2}$ = 20 min

FSH (R) In females \rightarrow Granulosa cells,
 In Males \rightarrow Sertoli cells (Spermatogenesis)

LH (R) In Females - Theca cells

Granulosa cells - appear only in
 Late proliferative phase

In Males - Leydig cells

↳ Produce Testosterone

FSH \Rightarrow i) do Selection of cohort of follicle every Month

(100)
(100)

ii) Selection & growth of dominant follicle,

iii) Ovulation → Final Release of ovum by collagen breakdown is brought about by FSH

(122)

LH → Function

- ii) Ovulation
- ii) Formation & Maintenance of corpus luteum
- iii) Final growth of Follicle

LH Surge $\xrightarrow[24-36 \text{ hrs}]{36 \text{ hrs}}$ Ovulation

LH Peak $\xrightarrow{12 \text{ hrs}}$ Ovulation

* LH Surge ⇒ Initiation by high level of Estrogen.
 $(200 \mu\text{g} \times 48 \text{ hrs})$

Maintenance of \Rightarrow E + P
LH Surge

Amount of estrogen to cause LH surge

* When does Progesterone Synthesis begins

↳ Before ovulation (36 hrs) $\xrightarrow{\text{Low in Amount}}$

LH → Surge → Luteinization of Granulosa cells

Q. Just before ovulation; which is true is

(A) ↑ LH; ↓ FSH

(B) ↑ FSH; ↓ LH

(C) Both ↑ (LH Peak >> FSH Peak)

(D) Both ↓

Small amount of Progesterone gives + feedback.

CORPUS LUTEUM \Rightarrow every Month \Rightarrow die

\hookrightarrow Life span - 14 days (constant luteal phase)

Q Q ib. 36 day Menstrual cycle; ovulation day ??

\Downarrow
on 2nd day

Q Q Which hormones maintain the corpus luteum

\hookrightarrow LH

Q Q Which hormones maintain the corpus luteum in ♀

\hookrightarrow HCG

Rescue the corpus luteum from luteolysis

Corpus Luteum

\hookrightarrow Progesterone

Estradiol

Relaxin

Inhibin A

Secreted by granulosa
cell of the follicle

Inhibin B

\curvearrowright

Inhibit the release of FSH

- Peak activity of Corpus Luteum \Rightarrow 8th day Post ovulation

\curvearrowright

Maxm Progesterone production

HYPOTHALAMUS → Release GnRH

GnRH — Arcuate Nucleus Released if (in Medial hypo-thalamus)

↳ decapeptide

$t_{1/2} = 3-4\text{ min}$

• Neurons (GnRH) derived → "Olfactory placode"

olfactory N.

Migration

Arcuate N.

Boys >> Girls

Absent Migration

"KALL MANN's Sx" (M/c Inheritance)

- No GnRH
- Anosmia
- No LH & FSH

X-linked Recessive

M/c gene involved

↳ KAL-1 gene

• GnRH Secretion in fetus

At 12 weeks

→

LH/FSH

Initially Tes up to 20 weeks

After

Suppressed

↓ child born

withdrawal of E/P

May born bleedit
PV; enlargement
of breast

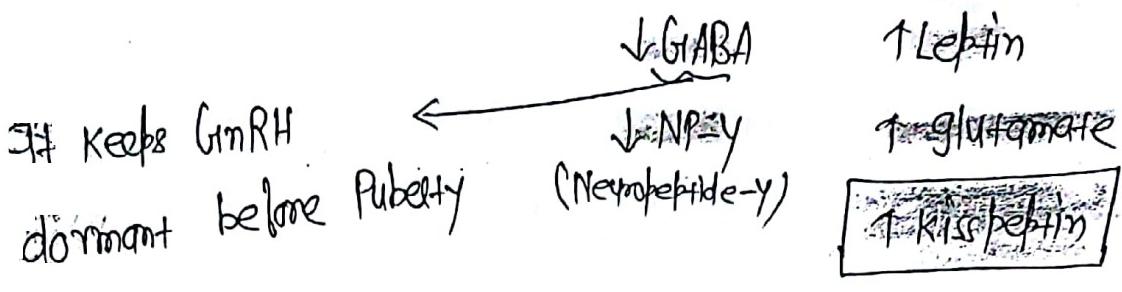
Sudden GnRH Tes

↳ LH/FSH (elevated Mo;
about 3-6
months after delivery)

Until Puberty

After this GnRH
Suppressed

* Activation of HP axis @ Puberty \Rightarrow ~~↓ Neurotransmitter~~



Pulsatile Release of GnRH \rightarrow Night time

Initial cycle May \leftarrow Release of LH \rightarrow @ Night time
be Anovulatory b/c
FSH Not Release; so;
Mature Follicle Not
Release

* GnRH Agonist \Rightarrow M/c used

\downarrow
Not effective orally.
give in two Manner

\hookrightarrow Lupronide
(Goserelin
Nafarelin
Buserelin)

Continuous

1st Response \Rightarrow ↑ LH|FSH

(flare response)

Persistently high (Inhibition HPO)

Pulsatile
(i/v Infusion pump)

Goserelin \rightarrow Delayed puberty
Ovulation induction

ContinuousEndometriosisFibroid UterusHirsutismPrecocious PubertyBreast CancerProstate CancerPulsatile

(24)

Kallmann's Syndrome

* GnRH Antagonist \rightarrow M/c used \Rightarrow Complex*

\downarrow No Flare Response (No GnRH ↑)

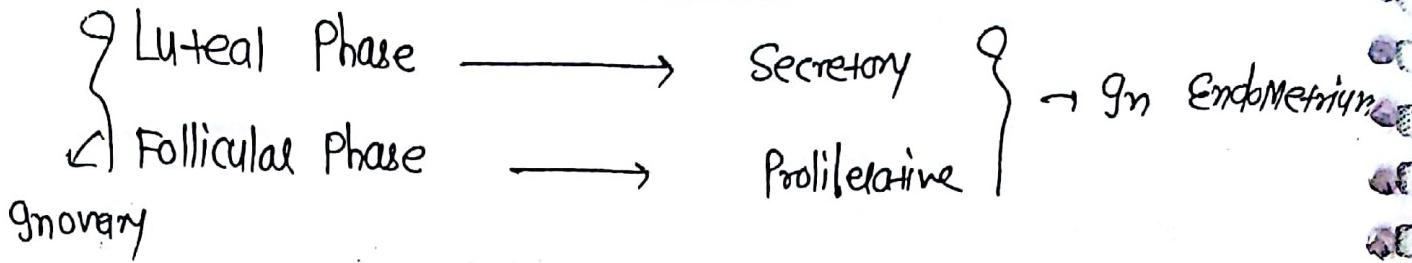
Somagon Antagonist - orally active

Indication \Rightarrow Same as continuous GnRH Agonist Indication

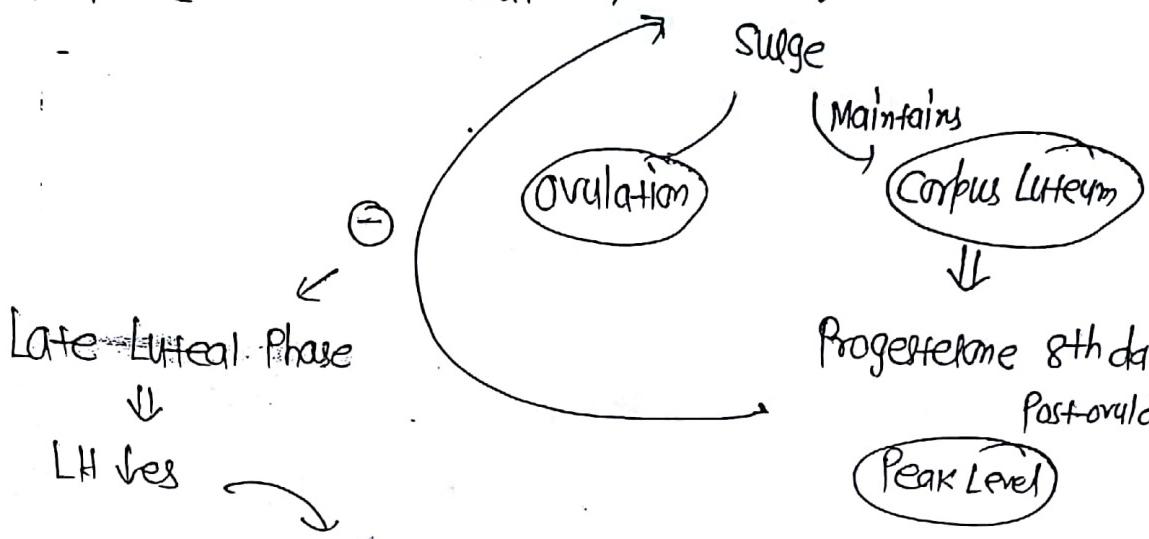
* GnRH \rightarrow LH \Rightarrow Pulse Frequency is high

\rightarrow FSH \Rightarrow Low Pulse Frequency

MENSTRUAL CYCLE



Luteal Phase → Start \Rightarrow onset of LH



Menstrual blood is mainly Arterial. Corpus Albicans

Spiral Arterioles

\uparrow vasoconstriction

Avascular \leftarrow Endometrium

Necrosis

\hookrightarrow sheds off (Menses)

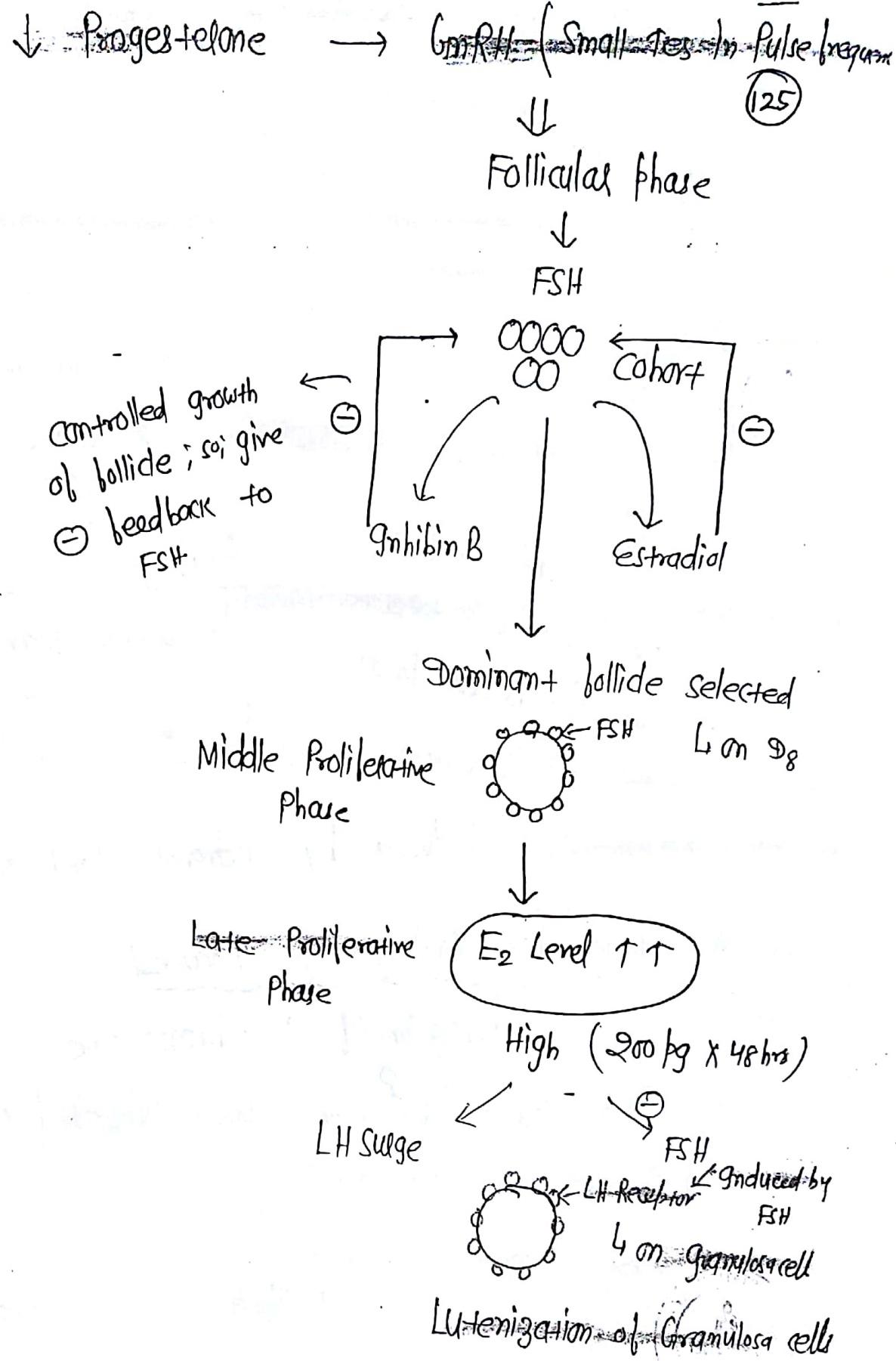
Myometrium

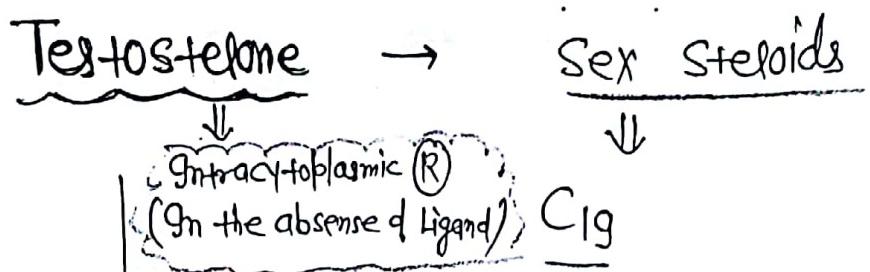


10 dysmenorrhoea (physiological) \rightarrow if present then ovulatory cycle \oplus

Anovulatory

bleeding \Rightarrow painless





Major Source ⇒ i) Peripheral Conversion of (50%)
of testosterone in ♀ Androstenedione

ii) 25% adrenal gland

iii) 25% ovary - theca cells

↓ (theca interna)

Androgen

↳ which androgen is in Max amount

Androstenedione > DHEA > Testosterone

- Ovary doesn't produce DHT (Dihydro Testosterone)
- Ovary also doesn't produce DHEAS sulphate

Produced by Adrenal gland only.

* Gene for Androgen (R) Located

↳ Long Arm of X-Chromosome

* Testosterone in ♀ ⇒ i) Pubarche / Adrenarche
↳ ^{dehydro Androgen} Adrenarche

* Estrogens

ii) Control of Libido

iii) ↑ Intraovarian Testosterone Level ↳

↳ Antral Follicular Growth
(Estrogen Rich Environment)

* Testosterone in ♂ → Spermatogenesis

(126)

Intra-testicular ↓ Level of testosterone is
↓ high

Sertoli cells produce TBP (Testosterone Binding Protein)

* ~~Initiation of spermatogenesis~~

↳ by FSH

* ~~Spermatogenesis Require~~ → FSH / Testosterone

* Sertoli cells produce → Mullerian Inhibiting substance

~~Testosterone binding protein~~

Relaxin

Inhibitin

Estradiol

* Most of Testosterone is in Bound form: Binds to SHBG / Albumin
↳ 1+ Testosterone free (Male 2% free).

* Testosterone → SHBG Synthesis

↳ takes place in Liver

Q: Which has higher affinity to bind to SHBG?

Testosterone → Estrogen.

* End product of Testosterone → ~~Excretion (ketonebids)~~

Q: Which cells form Blood-testis barrier

↳ Junction of Sertoli-Sertoli cells

Physical barrier b/w blood vessels & seminiferous tubules

2 compartments :

ADLUMINAL COMPARTMENT : 1° Spermatocyte

2° Spermatocyte

Inner side of tubules;
isolated from blood & lymph

Basal compartment : Spermatogonia

Outer side of tubule; In contact
with blood & lymph:

* Normal Menstrual cycle

↳ Length = 21-35 days

acc-to FIGO; Length = 24-38 days

Avg. Length \Rightarrow 28 days

Amount of blood loss \Rightarrow 80 ml

Average of blood loss (amount) \Rightarrow 35-50 cc

No. of days = 2-7 days

Average No. of days = $4\frac{1}{2}$ -5 days

* Abnormal Uterine bleeding (AUB) \Rightarrow

Menorrhagia \Rightarrow More (>80 ml) Amount or More (≥ 8 day) Menstrual day
Length of cycle = N

Hypomenorrhea \Rightarrow <2 day or <20 ml

Polymenorrhea \Rightarrow <21 day (<24) Length of Menstrual cycle

Oligomenorrhea \Rightarrow >35 day (>38) Length of Menstrual cycle

Metrorrhagia \Rightarrow Irregular bleeding / Intermenstrual

- classification of AUB acc. to FIGO \rightarrow

(127)

PALM - COEIN system \rightarrow

| | |
|-------------------------|-----------------------------------|
| AUB P - dl+ Polypl | AUB C - dl+ coagulation defect |
| AUB A - dl+ Adenomyosis | AUB O - dl+ ovulatory dysfunction |
| AUB L - dl+ Leiomyoma | AUB E - dl+ endometrial cause |
| AUB M - dl+ Malignancy | AUB I - dl+ Gastrogenic |
| | AUB N - Not yet classified |

M_x \Rightarrow acc. to its cause

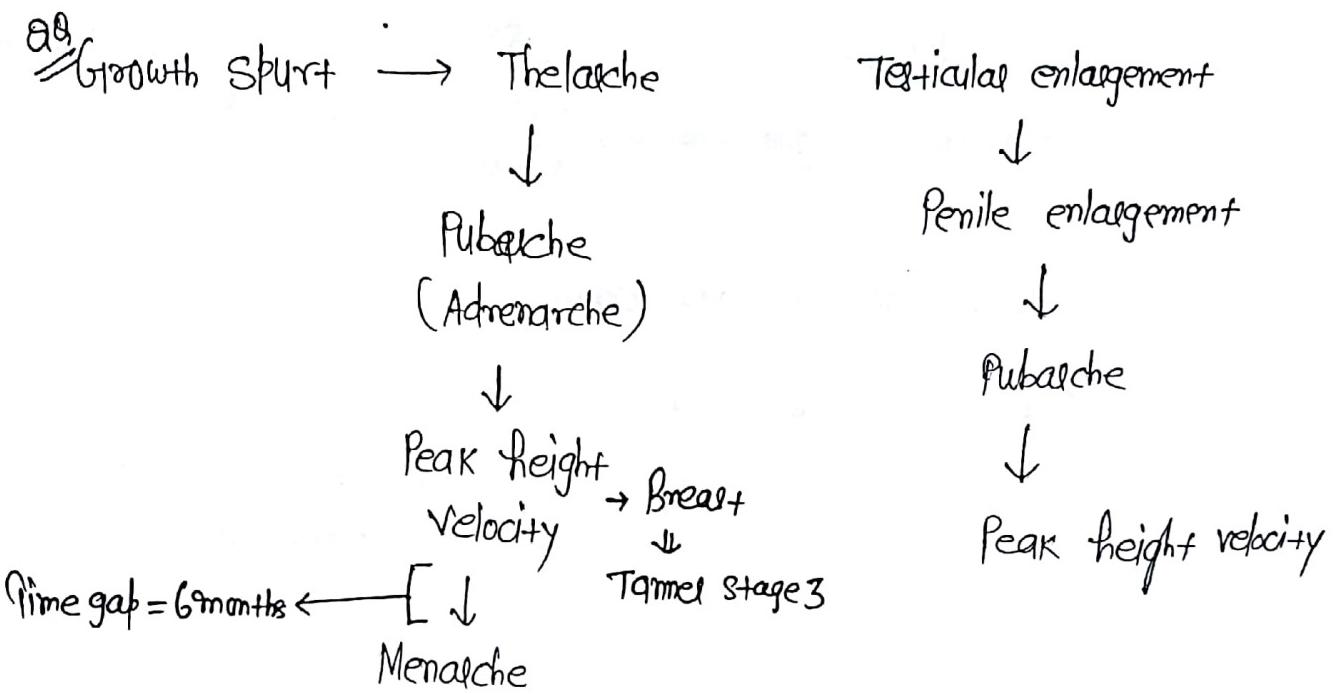
AUB ≥ 45 yr \rightarrow evaluated for Endometrial carcinoma
 (do Endometrial biopsy to Rule out Endometrial carcinoma)

PUBERTY

- development of secondary sexual character.

| <u>Girls</u> | <u>Boys</u> |
|--|---------------------|
| (N) Age \Rightarrow 10 $\frac{1}{2}$ yr | 11 $\frac{1}{2}$ yr |
| of Puberty | < 9 yr |
| Precocious \Rightarrow More common in girls < 8 yr | |
| Puberty | |
| Delayed \Rightarrow 13 yr | 14 yr |
| Puberty | |
| ↳ More common in boys | |

| | | |
|--|--|--|
| 1 st sign of Puberty | <u>Girls</u> Growth Spurt | <u>Boys</u> Testicular enlargement |
| 1 st visible sign of Puberty | Thelarche (Appearance of Breast bud) (Tanner stage - 2) | Testicular enlargement |



* M/c cause of Precocious Puberty in girls \Rightarrow Endocrinologic (90%)

so; girls come \in Precocious Puberty \leftarrow Brain tumor (10%)

\in MRI brain
evaluate

Brain tumor (10%)
M/c Brain tumor
 \downarrow
Hemangioma

Central Precocious

Puberty

- Premature activation of HPO axis

Peripheral Precocious

Puberty (128)

↓
Peripheral source of sex steroid hormones

↓
Estrogen / ~~Progesterone~~ Androgen

↳ secreting tumors

↓

M/c alw \Rightarrow McCune Albright syndrome
(Precocious Puberty +
Cafe-au-Lait + Polyostotic
fibrous dysplasia)

- LH/FSH ↑

↓

- Isosexual

↳ May be iso sexual or may be heterosexual.

- * M/c cause of delayed Puberty in Males \Rightarrow Constitutional delay

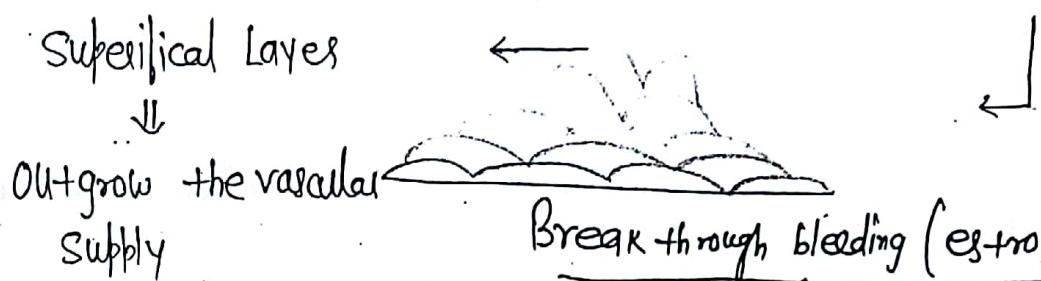
↓
Other relatives have same history of delayed puberty

- * Other problems in adolescent/ Pubertal age group

↳ Irregular bleeding - M/c cause Anovulatory bleeding

2nd M/c \Rightarrow coagulation defects

* In Anovulatory cycle → Unopposed estrogen



Breakthrough bleeding (estrogen)

↳ Necrosis → sheds off (More than N bleed)

↳ b/c endometrial is thick

* DOC ⇒ OCP

for Adolescent

Irregular bleeding

↳ Regulate the cycle

↳ also less blood loss.

Protects from Unwanted Pregnancy

also less Pelvic Inflammatory disease

also gives - only Progestrone pills

Q&A: 20 days bleeding → Unstable vitals

↳ So; endometrium is thinned at time of presentation

↓
firstly give high dose i/v estrogen

↳ do endometrium Proliferation quickly and stops bleeding by sealing the breaks

↓
finally give Progestrone only pills / combined ocp

* High dose estrogen only given in unstable vitals; blc in all other cases estrogen also causes closure of epiphyseal plate (stop growth spurt). 129

Least effective \Rightarrow Mefenamic Acid (NSAIDs)

\Downarrow
When hormonal therapy is not taken by girls.

Tranexamic acid (antifibrinolytic drug)

\hookrightarrow Stop bleeding

MENOPAUSE

- Cessation of Menses for 12 consecutive months.
- Avg. Age \Rightarrow 51 yr
- Avg. Age in India \Rightarrow 47 yr
- It doesn't need any investigation; it is history based diagnosis.

* Pre Menopause

(Premature ovarian failure

i^o ovarian insufficiency)

\Rightarrow < 40 yr

\hookrightarrow confirmed by SFSH Level; if it is $\geq 40 \text{ IU}$
on 2 occasions done 1 month apart \rightarrow diagnosed
Premature Menopause

* Delayed Menopause \Rightarrow doesn't happen by 55 yrs of age

\Downarrow
Endometrial Evaluation (endometrial biopsy)

FSH ↑

LH ↑

Estradiol → Yes ($< 20 \text{ pg}$)

Testosterone production from the ovary continues just like before

Gradual Process (climacteric phase)

↳ Hormonal changes start,
Perimenopausal Phase / early Menopause

M/c Symptom ⇒ Vaso Motor Symptom

↳ Hot flushes
↳ ↓ Estrogen withdrawal
↳ coincides w/ LH Surge

Hot Flushes

⇒ Sudden feeling of warmth followed by
Diaphoresis Last for 1-5 min.

↳ More @ Night, (so) disturb Sleep (wake cycle)

Moderate - Severe hot flushes disturb daily routine

↳ to Start HRT (Hormone Replacement Therapy)

Q: If patient has intact uterus ~~for hysterectomy~~

↳ give (E) + P → No role in Hot flushes
↳ Systemic therapy (transdermal > oral)

Q: In Post-hysterectomy patient

↳ only (E) (1st Line)

(130)

↓ if (E) is cl/I

give SSRI (2nd Line)

3rd Line drugs ↴

Clonidine

In pre-Menopausal

Gabapentin

Syndrome it is

Pregabalin

1st Line of drug

* if patient is Not tolerating oral Progesterone

↳ give Mirena (LNG-IUD)

(E) + Bezedoxifene (SERM + SERD)

protects the
Endometrium

good effect on
Bones

Other SERMs ↴

MC side effect
↳ Hot flush

Tamoxifene
Raloxifene
Clomiphene
Orymoxifene

Not used in Rx of
hot flush

Tamoxifene ↴ given in breast cancer patient
↑ Risk of Endometrial cancer

Raloxifene ↴ doesn't ↑ Risk of endometrial ca

↳ can be used for post-menopausal osteoporosis

* Doc for Post - Menopausal osteoporosis \Rightarrow Bisphosphonates

ORMELOXIFENE \Rightarrow Centchroman (Saheli)

↳ Indian government \Rightarrow Chhaya

Non-~~contraceptive~~ Steroidal contraceptive

It makes endometrium out of phase & prevents
Inflammation.

DUB (Dysfunctional Uterine bleeding)

PERTMENOPAUSAL WOMEN \Rightarrow Cycle become Anovulatory; so;

Irregular bleeding starts

(Estrogen break through)

doesn't bleed from 2 $\frac{1}{2}$ A

during Phase of
Amenorrhoea

gives OCP / P alone

during the phase of
active bleeding

43 yr q ; C 3 Month Amenorrhoea

Bleeding x 20 days

Unstable vitals

\downarrow

do D&C

Quickly stop
bleeding) in place
of high dose estrogen

Stable vitals

\downarrow

high dose oral

Estrogen \Rightarrow Stop the
bleeding in 24 hrs

\hookrightarrow after that give P/OCP

\hookrightarrow In Adolescent: no D&C b/c it alters the fertility

* Any Reproductive age women; comes \in H/o Amenorrhoea
 Firstly Rule out pregnancy.

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V.V.g. *** AMENORRHOEA

Primary

- Absence of Menstruation by 15 yr of age in the absence of 2° sexual character

OR

- Absence of Menstruation by 15 yr of age in the presence of 2° sexual character

M/c Cause \Rightarrow Gonadal dysgenesis

2nd M/c cause \Rightarrow Mullerian agenesis

Secondary

- Absence of Menstruation for 90 days in a previously menstruating female
 (In Irregular cycles $\varnothing \Rightarrow 6$ Months)
- Absence of Menstruation
- M/c Cause \Rightarrow Pregnancy
- M/c Pathological cause \Rightarrow PCOS

PRIMARY AMENORRHOEA

- Look about Thelarche

Absent

Present

↓

dt+ Gonadal dysgenesis $\varnothing \Rightarrow$ Gonads Abnormal

Kallman's syndrome

constitutional delay } \Rightarrow Gonads Normal

Gonadal dysgenesis (Raised LH; FSH)

- Turner
- Pure gonadal dysgenesis
- Mixed gonadal dysgenesis

TURNER

Karyotype \Rightarrow XO

Gonads — ovary —

PURE GONADAL

DYSGENESIS

$46\text{ XY} / 46\text{ XX}$

→ Swyer's Sx

(Mutation in SRY gene)

Non-functional

d/f absence of Y chromosome

Functional (SRY gene)

Internal
genitalia

Female —

absence of MIS / testosterone

(
Estrogen
 \downarrow → Absence
Growth Puberty \hookrightarrow Hypoplastic

External
genitalia

Female —

absence of DHT (active form of
testosterone)

Gonads
absence

↓
Ovary

↓
Accelerated Atresia

fibrosis (Fibrotic streaks)

Fibrotic gonads

↓
fibrotic ovary

Fibrotic / Nonfunctioning
Streaks / testes

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TURNER

Uterus \Rightarrow present
 (Hypoplastic)

External genitalia \Rightarrow Female
 Like

Pubic / Axillary hair \Rightarrow present
 (Sparse Scanty)

Breast development \Rightarrow Absent

Q.Q. Most characteristic feature of Turner Syndrome \rightarrow
 Short stature

Stature \Rightarrow Short stature
 $45\% O$
 ↓
 Absent \otimes
 ↓
 Absence of (SRY) gene
 ↓
 Responsible for growth
 of long bone

PURE GONADAL
DYSGENESIS

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present

MIXED GONADAL
DYSGENESIS

Mullerian / wolffian
 both ducts \oplus mif; b/c
 MIS act on i/L
 Mullerian duct

Female
 Like

Ambiguous genitalia
 (Like of both Male & female)

Present

Present

Absent

Absent

N/Fall

N/Fall

TURNER SYNDROME

- ↳ Short Stature
- ↳ Short Webbed Neck
- ↳ Low Posterior hair line
- M/c congenital heart disease \Rightarrow Bicuspid ^{Aortic valve} ~~heart~~
- ↳ Short 4th Metacarpel
- ↳ Cubitus Valgus (elbow deformity)
- ↳ Rudimentary ovaries
- ↳ No Menstruation



True about Turner's:

- (A) Uterus \oplus Breast \ominus
- (B) Both \oplus
- (C) Uterus \ominus Breast \oplus
- (D) Both \ominus

* I.Q. \Rightarrow N in Turner syndrome

I.Q. \Rightarrow Ab Sub Normal \Rightarrow when extra "X" chromosome

- ↳ In "Klinefelter Sx" (47XXY) \oplus
- ↳ M/c Sex chromosome Abnormality.

* Life-span \Rightarrow Slightly less

* LH/FSH \Rightarrow Yes (so; Turner Sx is class II Hypergonadotropic hypogonadism)

TURNER's (45X0)

FSH ↑

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(133)

KALLMAN'S (46XX)

FSH↓ (Hybogonadotropic
hypogonadism)

Short Stature

(N) / tall

Streak ovaries

(N) ovaries

Anosmia (−)

Anosmia (+)

Uterus (⊕)

(+)

Breast development (−)

(−)

External genitalia Female like

Female like

Pubic Axillary Sparse

Sparse

* constitutional delay

↳ Diagnosis of exclusion

Short (at) presentation

(N) Karyotype

* GOC for Primary Amenorrhoea \Rightarrow Karyotype

* Rx for Gonadal dysgenesis \Rightarrow give (E)/(P)

↳ She doesn't Need Surrogate Mother, Needs ovum donor

* Primary Amenorrhoea & Secondary sexual characters

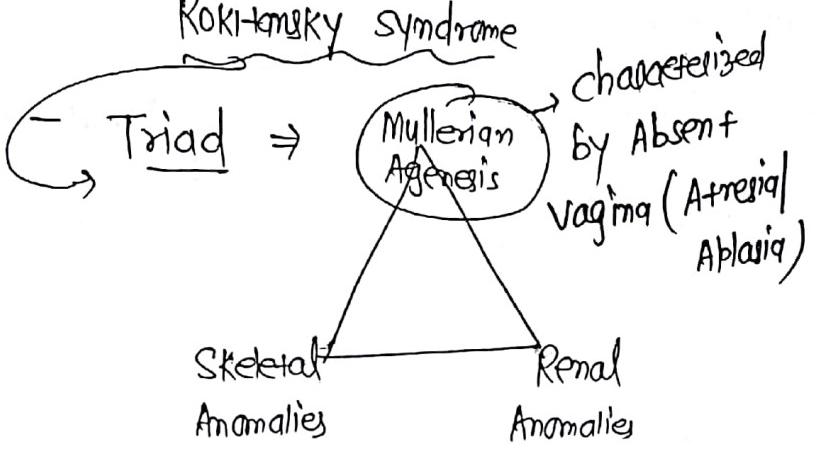


- i> Mullerian Agenesis;
- ii> AIS (Androgen Insensitivity Syndrome);
- iii> Imperforate hymen;
- iv> Transverse vaginal septum

Mullerian Agenesis

Klau "MRKH Syndrome"

Rokitansky syndrome



Absent vagina

Absent uterus

Fallopian tube are absent - Proximally
Present - Distally

- gonads - ovary (N)

Karyotype - 46 XX

AIS

- Klas "Testicular Feminization Syndrome"

- Testis is producing Androgen; but Receptor is completely insensitive

- Blind ending vagina (Small vaginal pouch)

- Uterus Absent (blk testis produce MII)

- Testis (f) (functional)
Karyotype \Rightarrow 46 XY

Mullerian Agenesis

AIS

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Breast development → Present

Present + @
Puberty

External Genitalia → Female Like

Female Like

Pt: May have inguinal hernia & carrying Undescended testis

↳ May cause Malignancy

do gonadectomy → after Puberty

if done before Puberty,

AIS patient Neither have female Nor
Male; so; done after Puberty

Male Pseudohermaphrodite

(M/c cause ⇒ AIS)

↳ Genotype Male; Phenotype Female

Female Pseudohermaphrodite

M/c cause ⇒ CAH

↳ deficiency of enzyme 21-hydroxylase

Female Phenotype → Y chromosome

↓

as soon as diagnosis is made ← Should undergo gonadectomy

Pubic /
Axillary hair

Mullerian Agenesis
(N)

AIS
Absent (Shave)
Scanty

Pregnancy

Needs Surrogate Mother
Generally - the child
is of Mullerian
Agenesis patient.
(by GVF technique)
- also vaginoplasty do

physically Absolutely
(N); but they
have worst Reproductive
outcome
↓
do vaginoplasty

Best to
differentiate \Rightarrow Karyotyping

S-testosterone
Level \Rightarrow (N)
(N female Level) Elevated
(Like of Male Level)

Q: After doing Karyotype in 10 Amenorrhoeas, Neg + test/
USG (\equiv)
USG + FSH (\equiv)

* TRUE HERMAPHRODITE \Rightarrow Tissue of both ovary & testes

[[also have Ambiguous Genitalia
]] Mosaic Karyotype

SD of Mixed gonadal dysgenesis \Rightarrow differentiate by
HPE of ovary & testis

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IMPERFORATE HYMEN

- Outflow tract obstruction
- everything else — Karyotype - (N)
 - Internal genitalia - (N)
 - External genitalia - (N)
 - development of Puberty - (N)
- H/o \Rightarrow Primary Amenorrhoea ; cyclical abdominal Pain
On Local examn \Rightarrow bluish distended Membrane
- Sometimes Prt. \in Emergency \Rightarrow Acute Urinary obstruction
 - HematoMeteor
 - Hematoctellos \Rightarrow Bladder outflow obstruction

TRANSVERSE VAGINAL SEPTUM

- M/c site \Rightarrow Upper 1/3rd of vagina
- Tf = cruciate incision — Imperforate hymen
 - Excision — vaginal septum (transverse)

* Rosenstein Syndrome \rightarrow Partial Androgen Insensitivity Syndrome

complete AIS \rightarrow clitoris - hypoplastic
Labia Majora - II

Partial AIS \rightarrow clitoromegaly seen
↳ Both girl looking or Boy looking Person May have Partial AIS (depend on how Much Receptors are Active)

* Precocious Menarche \rightarrow Menarche Starts ^{without} ~~before~~ \varnothing^o
↳ sexual character
↳ < 10yr Menarche Starts

SECONDARY AMENORRHOEA

Rule out \textcircled{F}

GOC! Hormone assessment

always together \leftarrow done \downarrow TSH \rightarrow both hypo & hyper thyroidism
Prolactin - Hyperprolactinemia

ble TRH is stimulatory to Prolactin.

FSH ($\pm LH / \pm E_2$) \leftarrow M/c cause
 \leftarrow M/c presentation \leftarrow ^{eg:} Pituitary Microadenoma ($< 10\text{mm}$)
 \hookrightarrow Ovarian & Amenorrhea

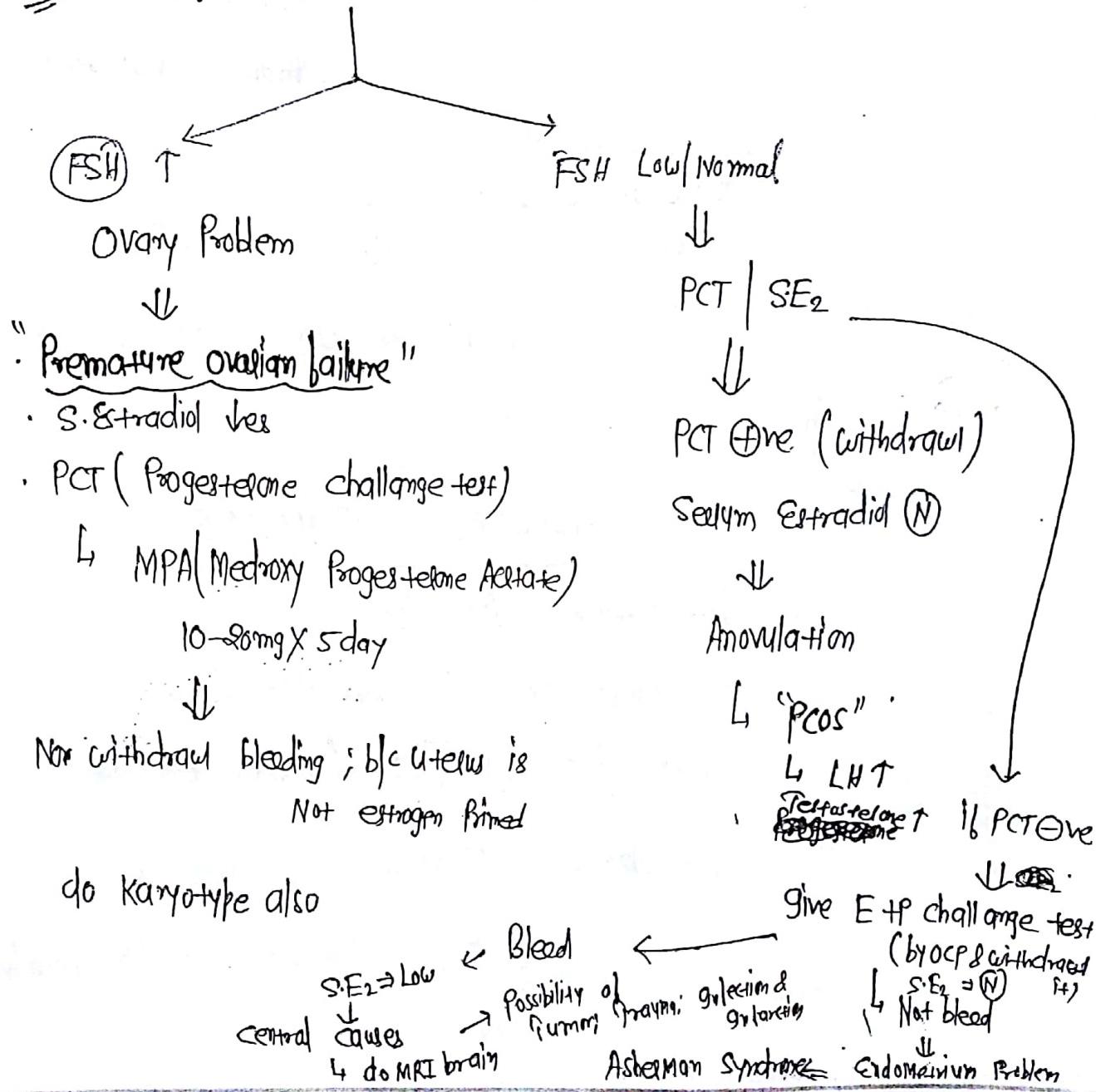
(136)

* Prolactin Over feedback Gm RH

↳ if $< 50 \Rightarrow$ Repeat test
 $> 50 \Rightarrow$ MRI

DOC for Prolactin \Rightarrow Cabergoline $>$ Bromocriptine
 (dopamine Agonists)
 Longer $t_{1/2}$; so give twice
 a week only.

Q&A if TSH & Prolactin \Rightarrow N



MRI brain



↳ (Functional hypothalamic Amenorrhea)

↓
Diagnosis of exclusion

↳ Seen in Anorexia Nervosa

Stress Induced Amenorrhea

Exercise Induced Amenorrhea

Chronic Malnutrition

Treatment of Premature ovarian failure

↳ HRT

(till the age of Menopause)

ASHERMAN'S SYNDROME

- Intrauterine Adhesions
- Mlc cause \Rightarrow Vigorous Curettage
- Highest Risk \Rightarrow PPH (vigorous curettage)
- Mlc presentation \Rightarrow Menstrual Irregularities
 - ↳ Amenorrhoea $>$ Hypomenorrhoea

* Single M/c Symptom/ Presentation

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↳ Infertility.

* Screening test \Rightarrow HSG (Not confirmatory test)

Adhesion seen as filling defect
in HSG

Multiple; Smooth;
Irregular Margin; Sma
in look.

IOC - Hysteroscopy (confirmatory test)
↳ both diagnostic & therapeutic

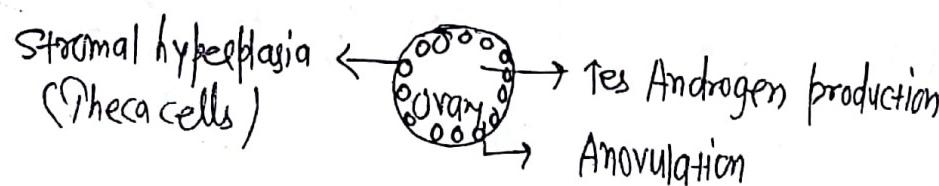
T/t \rightarrow

Hysteroscopic Adhesiolysis

+ CU-T Graftion + High dose Estrogen
to Make distance b/w
Uterine wall
for 1 cycle
for quickly Proliferation

PCOS (Polycystic ovarian Syndrome)

- Klas " Stein-Leventhal Syndrome" *
- Primary Pathology lies in the ovary.



- Tes Androgen production ; Not only inside ovary ;
but also in Periphery
 - ↳ Symptoms (Hirsutism)
 - also cause dyslipidemia
- 50% of girls have obesity (also produces excess Unopposed Estrogen)
 - ↳ Lib/c in Periphery
 - Androgen $\xrightarrow[\text{cell}]{\text{Aldo}} \text{Estrogen}$

- 75% of girls have Insulin Resistance

Syndrome X
(Metabolic syndrome)

- ↳ Hyperinsulinemia
- ↑ Androgen production
- dyslipidemia

↳ causes hyperinsulinemia

- ↑ Androgen production further
- dyslipidemia

* These patient have Proliferal Tes in LH

- ↳ Tes Androgen production

* HAIR AN → Acanthosis \oplus \Rightarrow Cutaneous Marker of Insulin Resistance

| | | | | |
|-------------------|--------------|--------------------|--------------|-----------|
| Hyper Androgenism | \downarrow | Insulin Resistance | \downarrow | Nigricans |
|-------------------|--------------|--------------------|--------------|-----------|

↳ Seen @ Nape of Neck
Axilla
Groin

Rotterdam's Criteria for diagnosis

If 2 or More of the following criteria are Met, provision diagnosis of PCOS;

i) Amenorrhea and/or oligomenorrhea
 ↳ diff Anovulation

* Menorrhagia ⇒ can be presentation (Mainly in obese patients)
 ↳ Not a criteria of Rotterdam's.

ii) Hyperandrogenemia and/or Hyperandrogenism
 ↓
 High blood Level
 ↳

Total S. testosterone ↑ (Mildly elevated) is Hirsutism → on chin; chest
 (N) < 70 ng/dl Axilla; thick hair growth

PCOS = 70-150 ng/dl
 definitely < 200 ng/dl

iii) Acne - Resistant to
 usual肥皂 and
 Scarring in Nature

if > 200 ng/dl (Severe) = Testostosterone
 Seizing Tumor

iii) Alopecia

* Scoring System to confirm the Hirsutism

↳ Ferriman Gallwey score ≥ 8

Not in PCOS

↳ Virilization (Hyperandrogenism)
 ↳ Severe testosterone - seen in Androgenoma

Virilization presents $\bar{c} \vdash$

- i) clitoromegaly
- ii) ↑ Muscle Mass
- iii) Hoarseness of voice
- iv) Male pattern balding
(Temporal Recession)

ii) USG Criteria of Rotterdam's

- a) ≥ 12 follicles in the ovary & each follicle $< 10\text{ mm}$ in size
And/or
- b) Ovary volume $\geq 10\text{ cc}$

* N Female ~~also~~ May have USG pictures of PCOS;
(20-25+)

PCOS Female may have Normal USG pictures

* if follicles seem large in USG \Rightarrow Hyperstimulatory ovary.
($> 1\text{ cm}$)

* On USG of PCOS \Rightarrow Necklace pattern
of ovary

No + Rotterdam's criteria

↳ also Stromal hyperplasia
Thick theca

↓
Characteristics to PCOS

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* If 2 criteria are Met; then Provisional diagnosis of \Rightarrow

P.C.O.S. >> Non-classical CAH (Adult onset CAH) (39)

1

Very common

Rae

To differentiate do \Rightarrow S. 17 OHP test (Screening test) $\xrightarrow{\text{Hydroxy Progesterone}}$

ACTH Stimulation (diagnostic test)
test

Other old of PCOS \Rightarrow Androgen tumor

↳ *giant Rabid onset*

Rapid progression

Testosterone >200

Virilization

Cushing syndrome

* Lab Investigation → i) S. testosterone
(DHEAS - May be slightly elevated
<700 Mgm)

11 > USG

1111 > LH↑ | FSH(N)

LH Ration \Rightarrow Yes ($> 2:1$)

IV T. Estragen T

$$\text{Total } E_1 \uparrow \quad | \quad TE_2 = N \quad | \quad \frac{E_2}{E_1} \text{ Ratio Reverse}$$

v) SHBG levels (so; Free E₂ Test)

↳ b/c testastetone inhibits SHBG Synthesis
↳ ↑ Hyperinsulinemia.

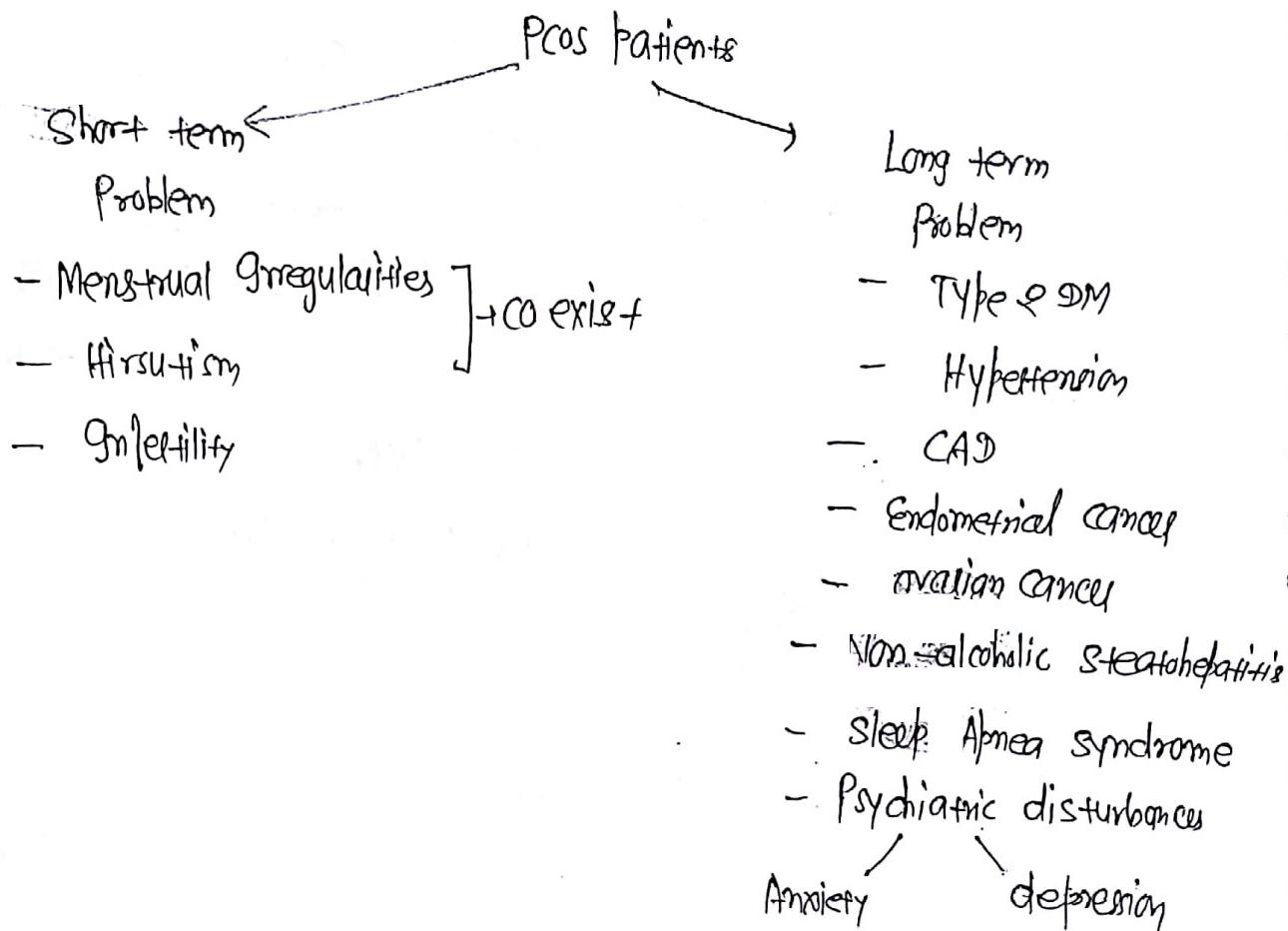
vi) Lipid Profile

vii) OGTT (to look for Insulin Resistance)

• Do $\frac{\text{Fasting Glucose}}{\text{Fasting Insulin}} < 4.5$

viii) TSH { N
ix) Prolactin }

x) 17 Hydroxy Progesterone test.



- Pregnancy Complication

↳ Abortion

Gestational DM

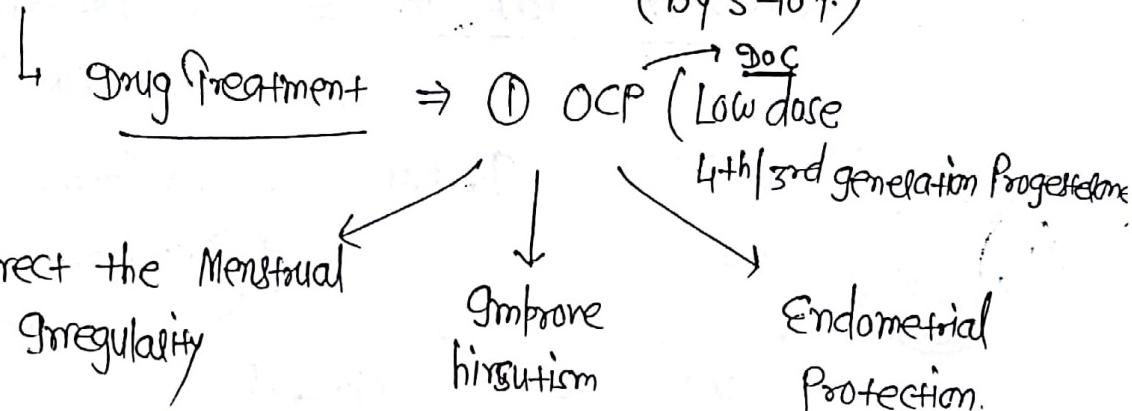
Pre-eclampsia

Pre-term Labour

Still-birth,

* Management → Menstrual Irregularities + Hirsutism

Obese PCOS Patient → Advice weight loss
(by 5-10%)



Goal for PCOS ⇒ OCP

* Only Hirsutism patient ⇒ give Spironolactone derivative

↳ Advice Not to conceive
b/c it is Teratogenic drug

*

Cyproterone Acetate

*

Flutamide

*

Finasteride

*

Ketoconazole

*

GnRH Agonist

*

Metformin

↳ Insulin sensitizers
weight loss (obese)

↳ Can cause Lactic Acidosis
do LFT / KFT

Eflornithine (topical)

all drugs are given in Hirsutism except \rightarrow Danazol
(Androgenics/E)

T/F for infertility \rightarrow d/f Anovulatory

Obese PCOS \rightarrow weight loss

(weight gain is also advice in less weight patient)

Ovulation induction \Rightarrow Clomiphene citrate
(SERM) does antagonistic activity

g+ has. ZG component
En component

MoA \Rightarrow Central

Estrogen Receptor on pituitary prevents feedback



Inhibition

FSH + (FSH/LH)

Starting dose 50mg D₂-D₆ / D₅-D₉ of Menstrual cycle

Maxm (Approved) drug \Rightarrow 100mg.

by FDA

Maxm approved times \Rightarrow 12 Months.

7. Women ovulate after clomiphene \Rightarrow 80% (141)
 of women conceive || \Rightarrow 40%

Antagonistic effect

- Cx Mucus \rightarrow Thick (Impenetrable)
- Endometrium - growth is affected

* Poor Response (Not ovulating)

Obese patient (Insulin Resistance) - Clomiphene citrate + Metformin (CC) also

ACOG obese PCOS patient \Rightarrow Doc for GnRH therapy

\hookrightarrow Letrozole (Not given for everyone; b/c
2.5mg \longrightarrow 7.5mg (Aromatase Inhibitor) ^{No FSA} approved,

* Clomiphene citrate \rightarrow Not teratogenic

MIC Side effect of clomiphene citrate \Rightarrow Hot flushes

2nd MIC || \Rightarrow Ovarian cyst formation

Other side effect || \Rightarrow Multifetal pregnancy

\hookrightarrow (6-8%)

\hookrightarrow Only twins

(Not higher order gestations)

\Rightarrow OHSS (ovarian hyperstimulation syndrome)

\hookrightarrow 1% (Negligible)

2nd Line drug for ovulation induction \Rightarrow

• Injection Gonadotropin (preferred)
(FSH/LH)

• Laparoscopic ovarian drilling \Rightarrow S/E
 \hookrightarrow Premature ovarian failure

• Gn.j. HMG
(Human Menopausal Gonadotropin)
 \hookrightarrow taken from Urine of Post-Menopausal women.

- Recombinant Purified \rightarrow Potent
preparation Gn.j. FSH

Highly expensive More S/E as well
 \hookrightarrow Multifetal - 3x higher
Pregnancy order Gestation
 \hookrightarrow OHSS - 15%

* Step up Protocol

Start \approx Low dose



Monitor Response

\hookrightarrow raising the dose.

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3rd Line drug for ovulation induction

Ind of ovulation induction
in hypothalamic cause / Kallmann's Syndrome

$\Rightarrow \frac{\text{GnRH}}{\downarrow}$

Pulsatile

* Multifetal \Rightarrow Gonadotropin > Clomiphene > GnRH Agonist
♀ Risk Cifrafe

* OHSS Risk \Rightarrow Gonadotropin > GnRH > Clomiphene > GnRH
Agonist Cifrafe Antagonist
 \downarrow
Prevention of OHSS.

In general \Rightarrow

Bromocriptine \rightarrow Anovulation

\downarrow diff hyperprolactinemia

Aromatase Inhibitors \rightarrow Letrozole

* OVARIAN HYPERSTIMULATION SYNDROME (OHSS)

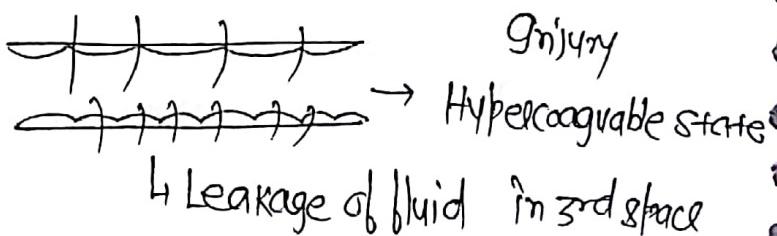
R/F \Rightarrow

- i) Younger Age;
- ii) PCOS;
- iii) High Serum E₂ Levels (>3500 pg)
- iv) Large size of follicle & Large No. of Follicles (>20 Follicles & >10 mm in size)
- v) Pregnancy
- vi) Gonadotropin

Causes \Rightarrow

Injection hCG (Used as ovulation Trigger);

• Mediator \Rightarrow VEGF \Rightarrow Cause Endothelial



Early - \rightarrow clin 9 days of inj. hCG

Late - \rightarrow 9 days of inj. hCG

↳ diff Pregnancy Late OHSS seen

Prevention \Rightarrow Delay the hCG Injection;

Called as "canceling" \leftarrow Cancel the cycle & do cryopreservation of embryo;

GnRH Antagonists

Volume expanders

Bromocriptine

INFERTILITY

- Inability to conceive with one year of Unprotected intercourse
- But; if the patient is > 35 yr \Rightarrow Inability to conceive even after 6 Months of Unprotected intercourse

Female factor $\xrightarrow{\text{contributes}}$ 40-55%

Male factor $\xrightarrow{\text{"}}$ 40%

Unexplained $\xrightarrow{\text{"}}$ 10%

- Female Factor \Rightarrow
 - Ovulatory Factor - 30-40%
 - Tubal Factor - 20-30%
 - Uterine Factor - 15%
 - Cervical Factor - 5%
 - Unexplained - 10%

↳ Ovulatory Factor \Rightarrow Most Reversible

↳ dlt Anovulation $\xrightarrow{\text{do}}$ ovulation induction

Premature ovarian failure $\xrightarrow{\text{do}}$ donor ovum

- Test of Ovulation \Rightarrow
 - i) Cervical Mucus
 - ii) vaginal cytology \rightarrow Lateral vaginal fornix (upper 1/3rd of lateral wall)
 - iii) Basal body temp. (Progesterone (BBT))
↳ Test BBT by $0.5-0.8^{\circ}\text{F}$
 - iv) we plot Bimodal graph - cycle is ovulatory

IV) easiest test \Rightarrow S. Progesterone



on D₂₁ \rightarrow $\geq 3\text{ ng} \Rightarrow$ ovulatory cycle

V) Best test - Endometrial Biopsy

↓

do in PreMenstrual phase-

2 days before the expected date of Menstruation.

If endometrium Report \rightarrow Secretory Endometrium

↳ ovulatory cycle

Proliferative Endometrium

↳ Anovulatory cycle, Telologic gland

↳ See Long tubular glands & Pseudo stratification

↳ d/t + estrogen hormone

While SubNuclear vacuolation \rightarrow is the evidence of Progesterone Secretion on D₁₆ on HPE

Cork-Screw glands \rightarrow

Seen on D₂₀ on HPE
(Late Secretory Phase)

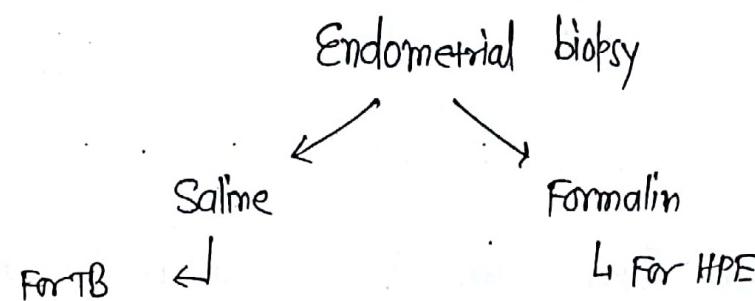
Max'm Stromal edema \rightarrow

Seen on D₂₂ on HPE

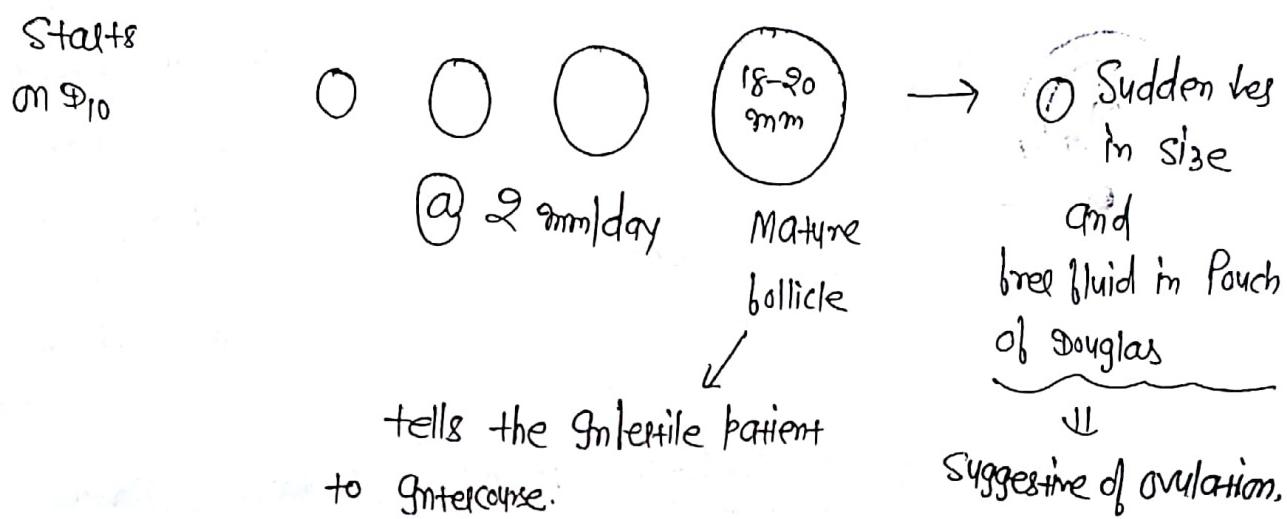
Leucocytic infiltration of Endometrium

(Premenstrual) on D₂₆ on HPE
Phase

* Endometrial biopsy at least once in evaluation of women infertility to rule out Genital TB; (144)



vii) M/c used test of ovulation in Infertile Patients TVs for follicular Monitoring ⇒



- also in endometrium on USG ⇒ Trilaminar Endometrium (Triple Layer Endometrium)

- ↳ Three hyperechoic lines seen in endometrium
- ↳ Seen in "Periovulatory Phase".

* Single hyperechoic line = Posterior Enhancement

↳ due to Secretion from Endometrium

Suggestive of Secretory phase

vii) Urinary LH kits

Urinary* LH Surge \rightarrow After 24 hrs
↳ ovulation takes place

OVARIAN RESERVE

M/c used test for it \Rightarrow i) S. FSH \Rightarrow D₂-D₄

↳ on D₃ (Best)

(N) value of S. FSH on D₃

↳ < 10 IU

10-15 IU \Rightarrow Borderline Reserve

> 15 IU = Poor Reserve

> 20 IU = Suggestive of Premature ovulation failure

> 40 IU \Rightarrow diagnosis of Premature ovulation failure

ii) S. Gnhibin B \Rightarrow on D₃ - < 45 pg - Poor Reserve

iii) AFC (Antral Follicle count) - on D₂-D₄

< 10 follicle \Rightarrow Poor Reserve

iv) Best test \Rightarrow S. AMH (Anti-Müllerian hormone; equivalent to MIS)

Very small amount - Small premenstrual follicle

< 0.5 ng - Poor Reserve

↳ b/c No fluctuation in menstrual cycle

(False test - less)

v) CCCT (clomiphene citrate challenge test)

$\text{D}_3 - \text{S.FSH}$

$\text{D}_5 - \text{D}_9$ - Clomiphene citrate 100 mg

$\text{D}_{10} \rightarrow \text{S.FSH}$

high basal level which rise further on D_{10} - poor Reserve

TUBAL FACTOR

Fallopian tube should be patent.

goc for patency \rightarrow HSG (screening test)

Best \rightarrow Laparoscopy + Chromoperfusion (Diagnostic)

Post-Menstrual Phase \rightarrow $\text{D}_5 - \text{D}_{11}$ day (M/c on D_{10})

\downarrow d/c + Cervix dilation

(+)nt @ this time

and also Pregnancy Ruled out @ this time

Peritoneal
spill

- cannula - Leech Wilkinson's cannula



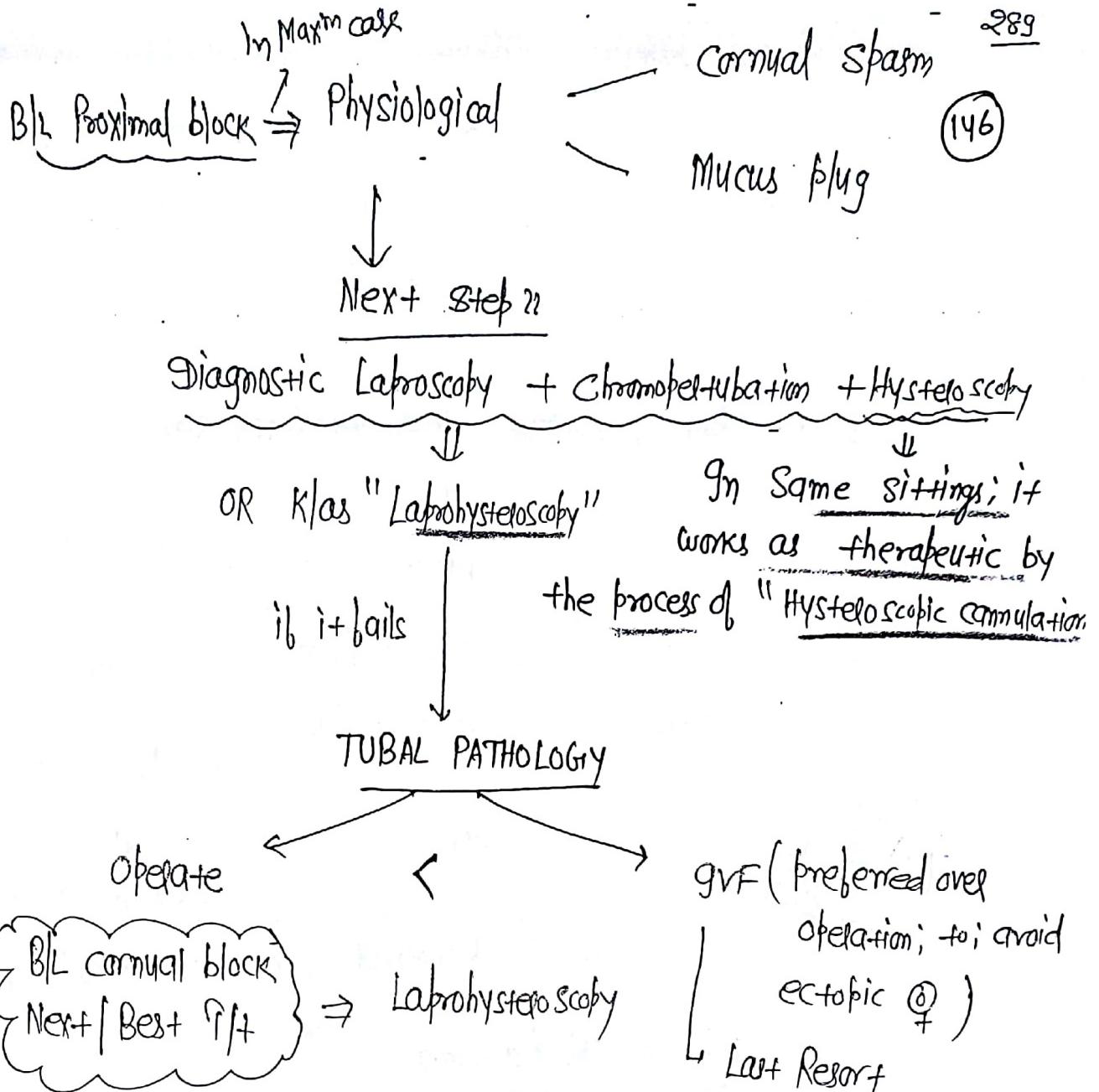
\downarrow
1mL dye (water-soluble Godinates
dye) wed

- * C/I of HSG \Rightarrow ① Suspected ♀ (UPT \rightarrow Ov^e);
 ② Known case of Genital TB;
 (Endometrial biopsy for Acid fast bacilli \rightarrow Ov^e)
 ③ Actively bleeding;
 ④ Current Pelvic Infection;
 ⑤ K/clo dye allergy

- HSG as screening for Uterine Pathologies
 - Adhesion
 - Polyp
 - Submucosal fibroid
 - Mullerian Anomalies

Other Screening Modality \Rightarrow Sono hysteroscopy
 ↳ Uses USG & Normal saline

- BL cornual block (Proximal block)
- BL Hydro salpinx \Rightarrow happen d+ distal block
 ↳ Represents severe injury
- BL Proximal block Mid segment block Distal block } \Rightarrow Best prognosis
 ↳ BL Proximal block
 ↳ very less pathologically



* If on HSG; B/L distal block seen

\downarrow
do Diagnostic Laparoscopy

Mild
 \downarrow ↑↑ by
Fibroplasty
Adhesiolysis

\rightarrow Severe (B/L hydrosalpinx)
 \downarrow
do gVF
 \downarrow
Fluid \rightarrow embryo-toxic
So; before gVF; do \Rightarrow
B/L Salpingectomy &
Cornual clipping.

* if on HSG; Proximal & distal block both seen on both of tubes separately (separately)



do gvf.

* if on HSG ; Mid segment-block seen



Tubal Ligation → Tubal Recanalization.

Good Prognostic Marker of conception
after Tubal Recanalization → Type of Ligation

Clips > Fallope Rings



Can be Reversed

Can't be Reversed ; cautery > Modified Pomeroy

ii) Type of Recanastomosis

Best ⇒ Gastro- Isthmic > Gastro- Ampullary

iii) Total Length after Recanastomosis > 4cm

iv) No other cause of Infertility

Test done before Anastomosis → Semen Analysis of husband

GENITAL TUBERCULOSIS

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147

- Secondary Infection;
- M/c Primary - Lungs > Lymph Node
- M/c Route -
 - Hematogenous
 - Direct
 - Lymphatics
 - Ascending Infections
- M/c Site \Rightarrow Fallopian tube > 90%
Endometrium 50-60%
- M/c Route \Rightarrow 
 - Direct
 - Spread
- U/c Site \Rightarrow Vagina & vulva - 12%
- Fallopian tube - Acute - Red Inflamed edematous
Chronic - Thick walled / Adhesions
- Cobble stone appearance on HSG
Tobacco Pouch appearance on HSG
Lead fibre or bite stem appearance on HSG
Beads on string appearance on HSG
Golf club appearance on HSG
B/L Hydro salpinx on HSG

* M/c site affected \Rightarrow Ampulla (1st site to be affected on Genital TB)

L/c site affected \Rightarrow Endometrial

Endometrium \rightarrow acute - Normal

\hookrightarrow affects only superficial part

Myometrium - spared

Chronic - Adhesion / Ulcers
(Asherman's Syndrome)

* M/c Presentation \Rightarrow Infertility (World over = 10%)
India = 17%)

- Pain

- Menstrual Irregularity

First Menstrual

Irregularity

\downarrow
Menorrhagia

More common

Menstrual Regularity

\downarrow
Amenorrhoea > oligo

* M/c Finding in Reproductive Age

\hookrightarrow (N) Pelvic exam

\hookrightarrow Tenderness sometime

L/c finding in Reproductive Age

\hookrightarrow B/L Adnexal Mass

B/L Adnexal \Rightarrow M/c finding in adolescent girls \in Genital TB 293
Mass

(148)

Diagnostic \Rightarrow \rightarrow Endometrial Biopsy (Best)

\rightarrow Menstrual blood PCR Analysis

(1st day sample taken to check superficial layer of endometrium)

Rx \Rightarrow ATT (Anti-tubercular therapy) x 6 Months

\hookrightarrow Improve fertility status - Yes.

* In Case there is severe Tubal Pathology
(distort Normal Anatomy)

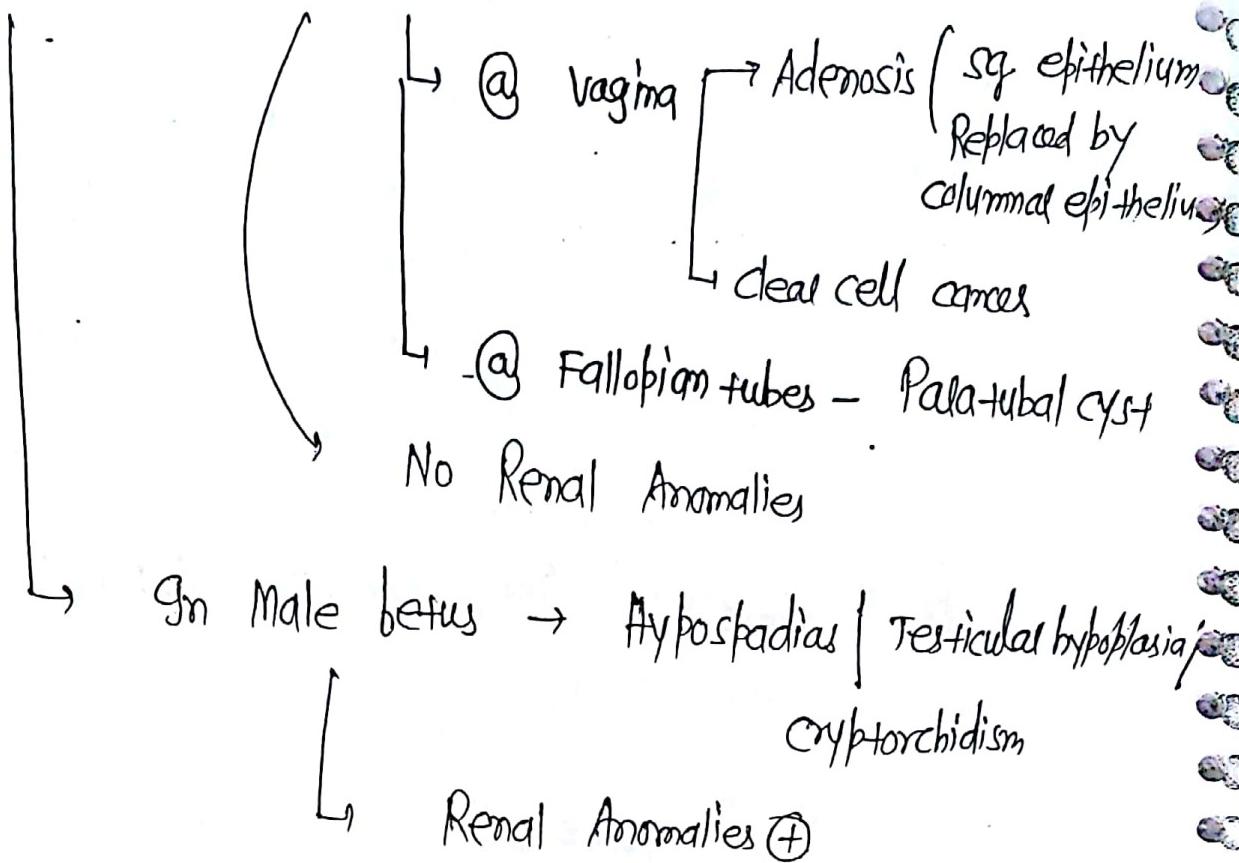
\hookrightarrow ATT doesn't improve fertility status;
go for IVF; but ATT is given
for Genital TB

UTERINE FACTORS

Infertility caused by \Rightarrow

- i> Fibroid - SubMucosal;
- ii> Polyp
- iii> Endometritis;
- iv> DES (Preg. women)
 - \hookrightarrow Fetus female fetus
 - \rightarrow Uterus (Hypoplastic Most characteristic T-shaped uterine cavity)

DES — In female fetus



V) Acutely Retroverted Uterus

Cochlear uterus ⇒ Acutely Anteverted Uterus

VI) Mullerian Anomalies

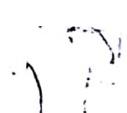
↳ M/c cause of Infertility — Septate Uterus

M/c cause of Abortion

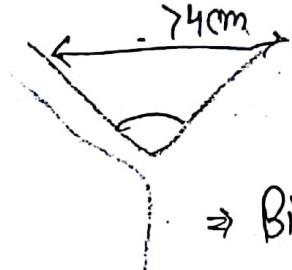
M/c Mullerian Anomaly

Worst Reproductive
Outcome

Best outcome ⇒ Arcuate > Endometrioid > Bicornuate



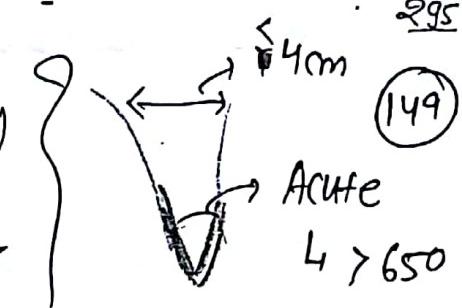
Screening test \Rightarrow



\Rightarrow Bicornuate

HSG
(Non-diagnostic)

Sebate
Uterus



$< 4\text{cm}$

149

Acute

> 650

Sign of sebate uterus on HSG

\hookrightarrow Bicornuate uterus

\Rightarrow Didelphius

IOC \Rightarrow MRI ~~+++~~ \Rightarrow if showing fundal dip $>1\text{cm}$

\Downarrow
Bicornuate

Sonohysterography

3D/4D USG (Not \supseteq USG)

\hookrightarrow usually do

Gold Standard Investigation

\Rightarrow ~~Laparoscopy~~ + hysteroscopy

\downarrow
alone is Not
a good Modality.

* All patients of Mullerian Anomalies Undergo evaluation
for \rightarrow Urinary tract anomalies (Renal USG / IVP)

\Downarrow
High Risk

* ectopic ovary \rightarrow ^{ab} Unicornuate uterus \rightarrow U/L dysmenorrhea
highest association C
Urinary tract anomalies

* TOC for Septate uterus

\hookrightarrow Hysteroscopic Resection
(Tompkins / Jones)

CERVICAL FACTORS

- Mucus affects fertility
- \hookrightarrow characteristics
 - Impermeable to sperm \rightarrow Klaus Sims Huhnel test
 - Anti Sperm Antibodies - Post coital test
 - \hookrightarrow Post coital Cx Mucus - Observed Under Microscope
 - Normal \rightarrow Forward Motility
 - ASA (+) - AbN Motility
 - circulatory
 - shaky
 - done @ 12-14th day of cycle

* Newel test \Rightarrow

Immuno bead Assay

Sperm Agglutination

Sperm Immobilization

ISO

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Not doing in
current clinical
practice.

* Tx of ASA \Rightarrow

i) Intrauterine insemination (IUI)

↳ Put Sperm directly into
Uterus; bypass the Cx Mucus

IOC for gynaecological infertility,

ii)

CC + IUI

↳ 6 Month

3 month CC alone

↳ 3 month CC + IUI

iii)

Mild Male factor
Infertility

Semen
Sample

Processing

Sperm Swim up

Sample (highly
lethal)

technique \downarrow use Membrane
Filter

iv)

Disorders of Sexual Intercourse

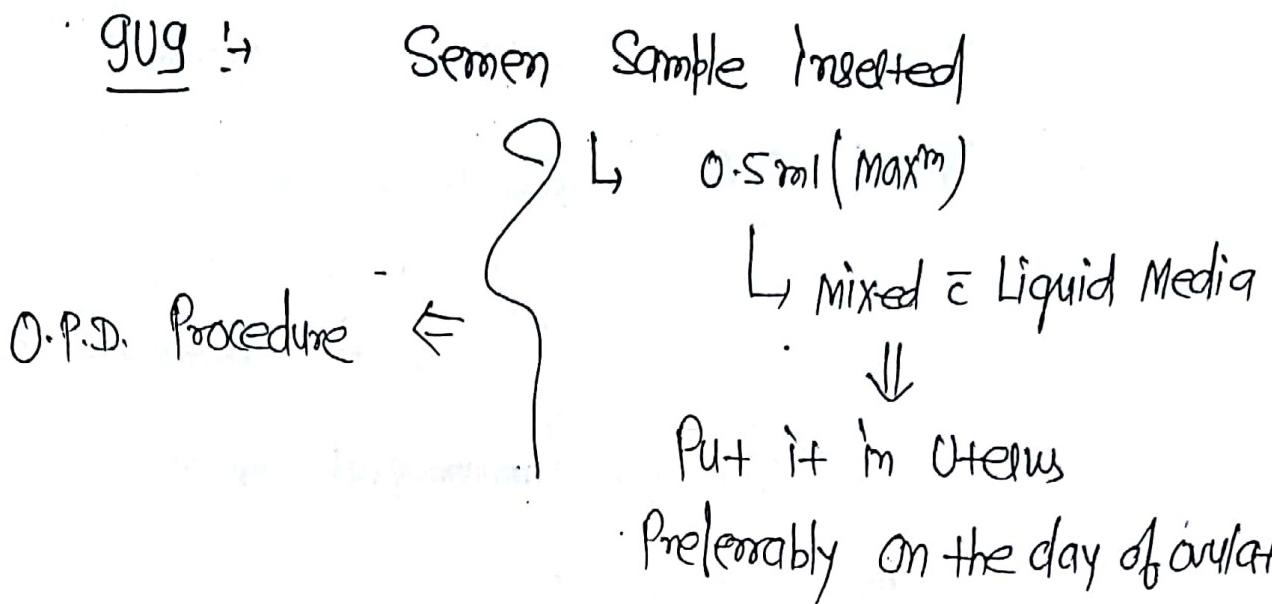
↳ Vaginismus

Failure of erection

Sexual

dysfunction

v) Absence of Male partner (Popular in developed world).



Tell's patient to reach the site of fertilization in
to be injected 10 minute
Position in Minimum
of 10 min.

Male Infertility Factors

MIC ⇒ defect in spermatogenesis

Semen Analysis ⇒ Best ⇒ Masturbation

Absstinence ⇒ 7d - 7day

Reach the Lab 6 hr 6 min
done after liquefaction

avg. time \Rightarrow 20 min

* WHO Parameters for Semen Analysis

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(151)

Lower Most Value → for fertile

Volume $> 1.5 \text{ mL}$

pH > 7.2

Total Sperm Count $\rightarrow > 39 \text{ million/mL ejaculate}$

($\text{N} > 100 \text{ million/mL ejaculate}$)

Sperm concⁿ $\rightarrow > 15 \text{ million/mL}$

Total Motility $\rightarrow > 40\%$

Progressive (Forward) $\rightarrow > 32\%$

Morphology $\rightarrow > 4\%$

Vitality $\rightarrow > 58\%$

WBC Count $\rightarrow < 1 \text{ million/mL}$

* Astermia \Rightarrow Absence of ejaculate

Azoospermia \Rightarrow Absence of sperms in ejaculate

Oligospermia $\Rightarrow < 15 \text{ million/mL}$

Asthenoasthma \Rightarrow (Abn) Motility

Teratospermia \Rightarrow (Abn) Morphology

Neospermia \Rightarrow Dead Sperms

Globospermia - Sperm - Rounded head
 ↓
 Lack Acrosomal cap

*

Azoospermia

↓ then 1stly do

- Confirmation on a 2nd sample

(1-4 week interval)



if azoospermia confirm



Do LH | FSH | Testosterone

if Non-obstructive case
 defective → Spermatogenesis



↑ LH | ↑ FSH | ↓ testosterone

• ↑ FSH | LH(N) | Teste(N)



Sertoli cells affected
 (Leydig cells N)

• LH↑ | Testosterone(N) / FSH(N)



Partial AIS

↳ Testosterone - N

FSH (Sertoli cell - FSH↑)

"Klars" Obstructive Azoospermia ⇒ obstruction in Semen Pathway

→ if site of ejaculation block vas deferens

⇒ Scrotal USG

→ if site of ejaculation block on ED (volume ↓)

⇒ Trans-Rectal USG

↓
 dilated seminal vesicle
 → absent seminal vesicle
 ↓
 cystic fibrosis (CFTR-gene)
 mutation

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* If Testes Involve \Rightarrow 1^o Hypogonadism
 (Hypergonadotropic) (152)

~~Testosterone~~

* Non-obstructive Azoospermia

do for Non-obstructive Azoospermia cases

\downarrow Microsurgical TESE (Testicular sperm extraction)
 \downarrow ICSI (Intracytoplasmic sperm injection)

* If Sperm count (15 million/mL) $\xrightarrow{\text{do}}$ GUG
 ($5-15 \text{ million/mL}$) $\xrightarrow{\text{do}}$ GVF
 ($<5 \text{ million/mL}$) $\xrightarrow{\text{do}}$ GCSI

* ~~Sperm concn + Motility~~ \Rightarrow Morphology (~~Most imp.~~ Parameter in N sperm)

* ART \Rightarrow Simplest form \Rightarrow GUG

All are ART except \Rightarrow ~~GUG~~
 ZIFT
 GIFT
 GCSI
 GVF

GVF

- do for -
 - i) Tubal factor infertility
 - ii) Male factor infertility
 - iii) Unexplained infertility

↓

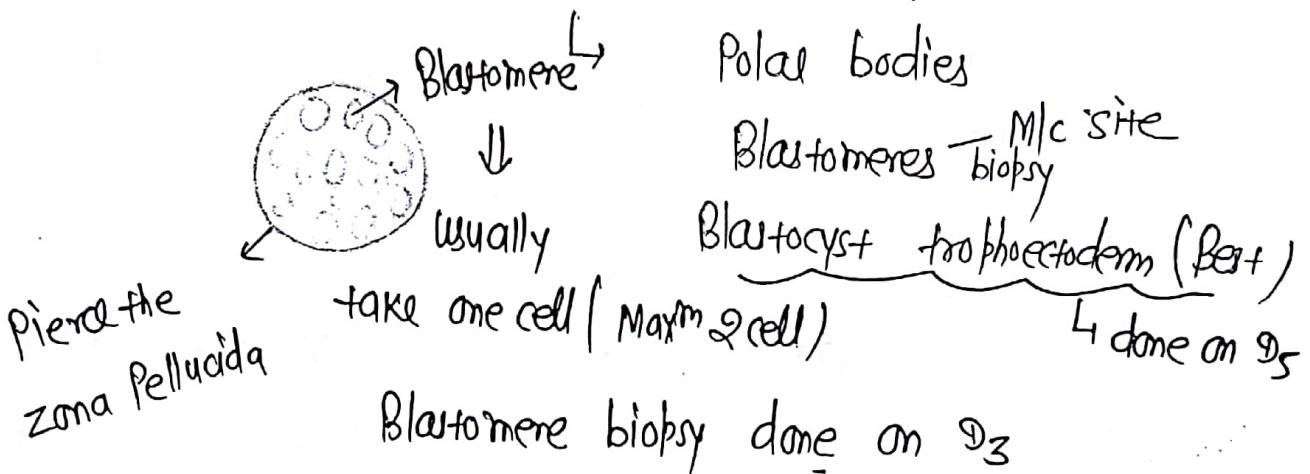
give 1st clomiphene citrate + gug X 3 cycles

for superovulation; Not
for ovulation induction ↓ if don't conceive
do gvf

IV) PIGD (Preimplantation genetic diagnosis)

↓

Site of Genetic Material taken



V) Premature ovarian failure

- Parts of GVF → 1st ovulation induction

↓ by inj. Gonadotropins

Follicular grow (Monitor growth)
↓ by Number, size; S.E.

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↓
Ovulation triggered by inj. HCG

↓ 34-36 hr after

Ovary pick-up (under USG guidance)

↓ on same day

GVF (test-tube Embryo formation)

↓

do Embryo transfer (M/c on D3).

↳ Never Put one embryo; Minⁿ-2
Embryo Put & at 2 cm below fundus
Under USG guidance

ENDOMETRIOSIS

- Presence of Endometrial glands & stroma outside the Uterus.
- M/c site \Rightarrow Ovary $>$ Pouch of Douglas $>$ Posterior leaf of broad Ligament $>$ Uterosacral Ligament $>$ Fallopian tube
- Occur at all sites except \Rightarrow Spleen.
- dependent on Estrogen for growth (ovarian steroids)
 ↳ so; disease of Reproductive age grp (25-35 yr)

- It is rare in adolescent & perimenopausal/postmenopausal women

- In Pregnancy — Maxm time endometrium
↳ blc of continuous — Progesterone Rich condⁿ
 ↳ Regress
 ↓
 decidualization

PGEI

Theory of endometriosis

↳ Most accepted theory

↳ Retrograde Menstruation
(Sampson's theory)

2nd Most accepted theory

↳ Coelomic Metaplasia

3rd ⇒ Immune Mediated theory

↓
Women w/ endometriosis have deficient cell
Mediated / humoral immunity

4th ⇒ Genetic theory ⇒ K-ray

if one first degree Relative affected

↳ chance of endometriosis is 7 times more

(154)

5th ⇒ Lymphatic / hematogenous



Umbilical Endometriosis

↳ Lymphatic explaining it very well

DOC ⇒

Diagnostic Laparoscopy

confirm the diagnosis

(Stage)

Biopsy (HPE)

↳ if altered coloured blood +

↳ chocolate cyst ⇒ frt. in ovary

↳ Red flame lesion (New lesion)
of endometriosis

↳ Powder burnt lesion (chronic lesion of endometriosis)

Minimal → Superficial isolated implants

Mild → Superficial + Multiple
aggregates diameter < 5cm

Moderate → Superficial + deep lesion
(> 5mm deep)

Severe → If endometriosis distorts Pelvic Anatomy
↳ dense adhesion
↳ chocolate cyst

On USG \Rightarrow Chocolate cyst can be pickup

↳ Homogenous Ground glass appearance

On MRI \Rightarrow only pick up the chocolate cyst

↳ Adolescent girl comes \equiv Endometriosis.

"Mushroom gap sign" seen.

* CA-125 \Rightarrow Raised in Endometriosis

↓
Normal < 35

↓
In Endometrioma \Rightarrow CA125 > 100
(Rupture)

24/5/18 Presentation \Rightarrow Pain + Adnexal Mass + Infertility.
(M/c)

Menstrual complain — Menorrhagia
Bowel / Bladder symptoms \oplus

Catamenial Hemo-thorax / Pneumothorax
↳ @ the time of Menses (May be endometriosis goes to Lung)

PAIN \Rightarrow M/c \Rightarrow Dysmenorrhoea $>$ Chronic Pelvic $>$ Dyspareunia $>$ Low Back Pain

↳ 2^o dysmenorrhoea

[10 dysmenorrhoea

dt+ Progesterone withdrawal

2^o dysmenorrhoea

dt+ Underlying disease process
(Endometriosis)

RTO

1^o dysmenorrhoea
Spasmodic

center (Suprapubic)

begins Mostly on the 1st day of Menstruation

Gm factors \subset blood flow
 \subset clin 72 hrs - Relief

Rx \Rightarrow Very Responsive to NSAIDs

2^o dysmenorrhoea 30%
Congestive (SS)

Localised (on one side)

PreMenstrual
(week before Menstruation)

doesn't improve \subset flow;
persist even Post Menstrual

Less Responsive to NSAIDs
* also has Dyspareunia;

{ Rectovaginal Septum;
deep endometriosis

Why Pain in endometriosis \Rightarrow

Gm implants \rightarrow [Blood] \rightarrow Inflammatory cells
 \downarrow Release

Inflammatory Mediators

Sign of chronic inflammation
cause of pain \hookrightarrow healed by Fibrosis

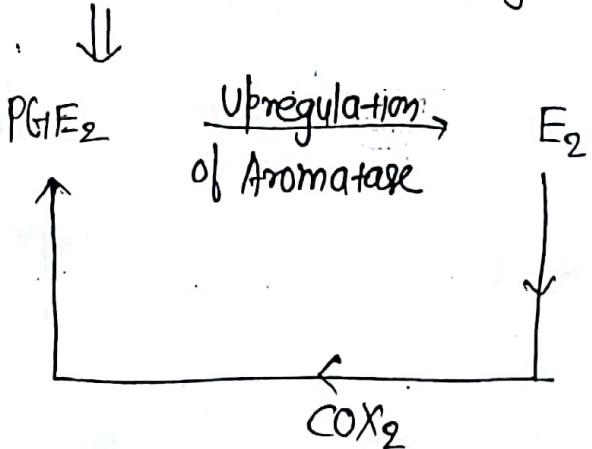
\hookrightarrow Cause Adhesion

\hookrightarrow also cause pain

* Pain in - Gm implants
 \Downarrow

Neuromodulation (\uparrow Nerve Endings - Pain sensation)
 \hookrightarrow cause by Estrogen,

* Implants \rightarrow Estrogen is in Permanent Excess state



(N) Endometrium doesn't have excess Aromatase for Upregulation of Aromatase

\downarrow
Eutopic

(N) Endometrium has 17- β OH dehydrogenase; which Metabolizes E₂.

* What we do for Pain in Endometriosis \Rightarrow

Pain (Suggestive of endometriosis)

Nature of Pain??

Minimal - Moderate

Severe Pain

\downarrow Reduce the
OCP's + NSAIDS Mediators

\downarrow Give GnRH Agonist

(given after diagnostic Laproscopy)

\downarrow & Suppress the
Ovary

If she wants to conceive
then only NSAIDS given

Dienogest (Latest Progestrone)

& oral MPA

DMPA (Inj drug; works for 3 months)

Mirena (LNG-IUD)

\downarrow No Improvement

\hookrightarrow Give Progestrone (do decidualization)

(156)

if After Progestrone female
is Not Responsive

↓
Give GnRH Agonist. (given after confirmation of endometriosis)

↓
by Laparoscopy
Given in Continuous Manner

Can also Give GnRH Antagonist.

↓ if Not Responsive
Give Danazol → higher Androgen S/E

Aromatase inhibitor → it can produce hypoestrogenic state

→ if given for More than six Months ⇒ Result in Bone Loss;

So; given " ADD - BACK THERAPY " ⇒ Given NORETHINDRONE

↓
to protect bone loss.

- * Surgical Management of Pain ⇒
 - i) Adhesiolysis;
 - ii) Fulguration of Sympathetic;
 - iii) LUNA (Laparoscopic Uterosacral Nerve Ablation);
 - iv) Presacral Neurectomy
 - ↳ Not effective;
Not done Routinely.

v) Adrenal Mass

↳ Endo Metrioma ⇒ Not Responsive to Medical Rx after

* TxOC for Endometrioma

↳ Cystectomy

I & D of the cyst → Recurrence Rate
(destroy the cyst wall) (80+)

if cyst is $< 5\text{cm}$ → wait & watch

if cyst is $> 5\text{cm}$ → do cystectomy.

Hysterectomy (Last Resort)

↳ only in case of family is complete

ADNEXAL MASS

P/V examination : findings who tells about endometriosis \Rightarrow

i) Tenderness in Pouch of Douglas;

ii) tender Nodules on Uterosacral Ligament;

iii) Fixed Retroverted uterus

iv) Adnexal tenderness;

v) Adnexal Mass



Ground glass appearance on USG

Infertility in Endometriosis \Rightarrow Main Reason \Rightarrow Ovarian (Not Anovulatory) (157)

- Folliculogenesis \Rightarrow defective
- Genetic Material \Rightarrow Not good of ovum

In Moderate - Severe endometriosis \Rightarrow Main Reason \Rightarrow Ovarian + Tubal

In Minimal - Mild Endometriosis \Rightarrow Rx Clomiphene Citrate + Intrauterine insemination X 3 cycle (3 months)

\downarrow
Superovulation

\downarrow
don't conceive

\hookrightarrow do GVF

In Moderate - Severe Endometriosis \Rightarrow Rx GVF + Sx
 \hookrightarrow do Mainly; Surgery is Not Necessary to do.

* GnRH Agonists has No Role in Improvement of Infertility in endometriosis

* Endometrioma \Rightarrow If during the GVF, we find the chocolate cyst

Wait & watch

\downarrow
don't operate: b/c if operate, b/c of follicle damage; infertility may seen.

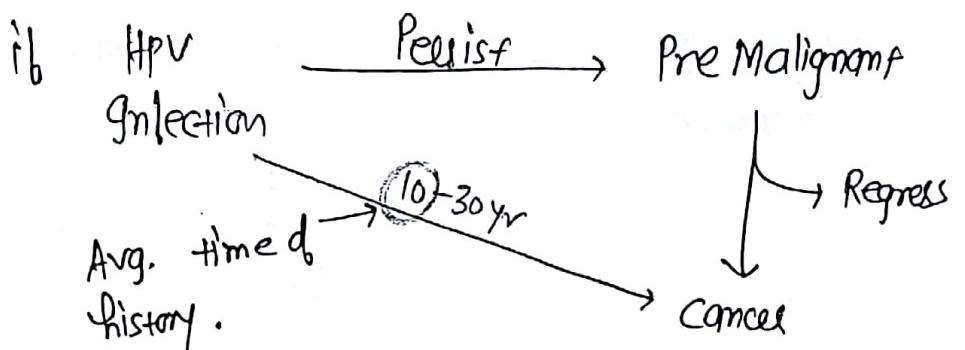
* MALIGNANCIES

* M/c Cancer Among Indian Women \Rightarrow Breast Cancer
2nd " " " " " \Rightarrow Cervical cancer

M/c cause of Mortality due to cancer in Indian women \Rightarrow "

CERVICAL CANCER

Very commonly a/w HPV infection (Most women clear this infection)



If all history is given do "Universal Screening"

- Acc. to ACS/ FIGO

Starts Screening \Rightarrow 21 yrs (irrespective of sexual activity)
In

How \Rightarrow PAPs \rightarrow 3 yearly in 21-25yr; everyone will turn out +ve;

Higher specificity \Rightarrow PAPs + HPV (co-test) \rightarrow 5 yearly so: Not do. Higher sensitivity (ideal ≥ 30 yr can do in ≥ 25 yr)

Stop \Rightarrow @ 65yr (provided the PAPs in last decade \textcircled{N})
if Not Normal Stop @ 75yr.

WHO Programme

Start of Screening

⇒ begins @ ≥ 30 yr

3rd - 4th decade } More targeted
(30-49 yrs) } Population Age Groups.



WHO says "SEE & TREAT".

How!! ⇒

do single test

VIA HPV > VIA \geq PAPs

\equiv (visual inspection

\subset Acetic acid)

\downarrow

ideal

\downarrow for confirmation

Best Method ⇒ HPV + VIA

if HPV alone done \Rightarrow 5 yearly do

if VIA & PAPs done \Rightarrow 3-5 yearly do

Treatment Protocol alc + WHO \Rightarrow do cryotherapy \circlearrowleft LEEP > conization
if Not eligible goes for LEEP

* Screening Methods \Rightarrow HPV testing (DNA testing)

• For high-risk viruses

\downarrow do via Hybrid capture Technique

\downarrow expensive

- if HPV + PAPs \rightarrow cotest
- if do PAPs 1st then follow up by HPV \Rightarrow reflex testing

* High Risk viruses

↳ HPV 16, 18, 31, 33, 35, 45, 52, 56, 58
 ↓ ↓
 70% of cervical 30% of cervical
 cancer cancer

* Routinely in India we do \Rightarrow VIA (visual inspection \in Acetic acid 5%)

↓
 Abnormal \rightarrow White area (dysplastic cells \oplus)
 ↓
 Aceto white area (stained Tcs)
 Unstained Area \Rightarrow Normal

VILI test — Lugol's iodine - 4-5%.

Klaas "Schiller's test"
 ↓ ↓
 Brown — \oplus test Normal cells \in Glycogen
 Unstained (Abnormal test) \oplus in mature cells
 ↓ blc of \ominus ce of dysplastic cells (immature cells)

• PAP Smear — Screening test (highly specificity)

↳ Taken by "Ayer's spatula & Cytobrush".

↓
 PAP Smear is always taken from ^{taken secretion from endocervix secretion} Bilid end of it & turn it by 360° . (2)

Taken from Transformation zone $>$ Squamo columnar Junction $>$ Ectocervix
 of cervix,

(159)

* Smear taken on ONE SLIDE

No Air drying do



AIIMS Nov'17

Fixative - ? ~~95% Ethyl alcohol +~~
5% Ether

* No Absolute Contraindication for PAP Smear; but if actively bleeding patient comes then tell her to come after bleeding stops.

* PAP Smear is also known "Secretion Cytology".

* BETHESDA CLASSIFICATION ⇒

① Normal Report ↳ tells about Report comes on PAP Smear.

② Reactive & Reparative changes (Healing Inflammatory changes);

③ Infection — Specify the organism

- * Organism lies inside endocx; tough to catch in PAP Smear ⇒ Chlamydia
Gonorrhoea

④ ASCUS — Atypical squamous cells of Undetermined significance
ASC-US (Modified Bethesda classification)

⑤ LSIL — Low grade squamous Intraepithelial Lesion

⑥ HSIL — High " "

⑦ Cancer

*

LSIL

HSIL

Nucleus
Cytoplasm



No. of cells

Less

More

Granules

evenly distributed

clumps

(Granular chromatin)

Membrane

Shrivelled Membrane Doubt

Ideally



PAPs
done

→ For Confirmation



Biopsy done

(For looking Abn Area)



Use Colpo Scope → Focal Length = 30
Magm Magnification = 30



Qib PAPs comes
abnormal

Colposcopic directed Biopsy

We can see Ectox

vaginal wall

vulva;

but we can't see Endox

* Abnormal Area from where Biopsy should be taken ³¹⁷ \Rightarrow

i) Irregular Surface contour

(160)

ii) Mucosa - Pale in colour

iii) In Acetic acid \rightarrow White area seen

iv) In vascular pattern of



Reticular

Mosaic

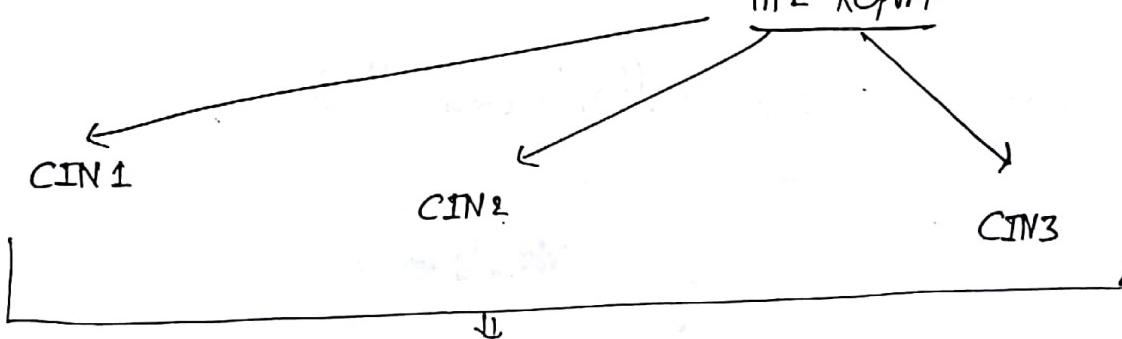
Punctate

To visualise vascular pattern

clearly; we add Green filter to colposcope

* Since it is Biopsy; so; Colposcope Report called as

"HPE Report"



all are Pre Malignant cond'n i.e all have basement

* HPV cells affects basal & parabasal cells; so, Membrane intact.
Cancer starts from Lower part of epithelium

* Dysplastic cells - all Seen in Lower 1/3 of the epithelium

\hookrightarrow CIN1 \Rightarrow Mild dysplasia

\hookrightarrow LSIL = CIN1 = Mild dysplasia

CIN2, CIN3 = HSIL

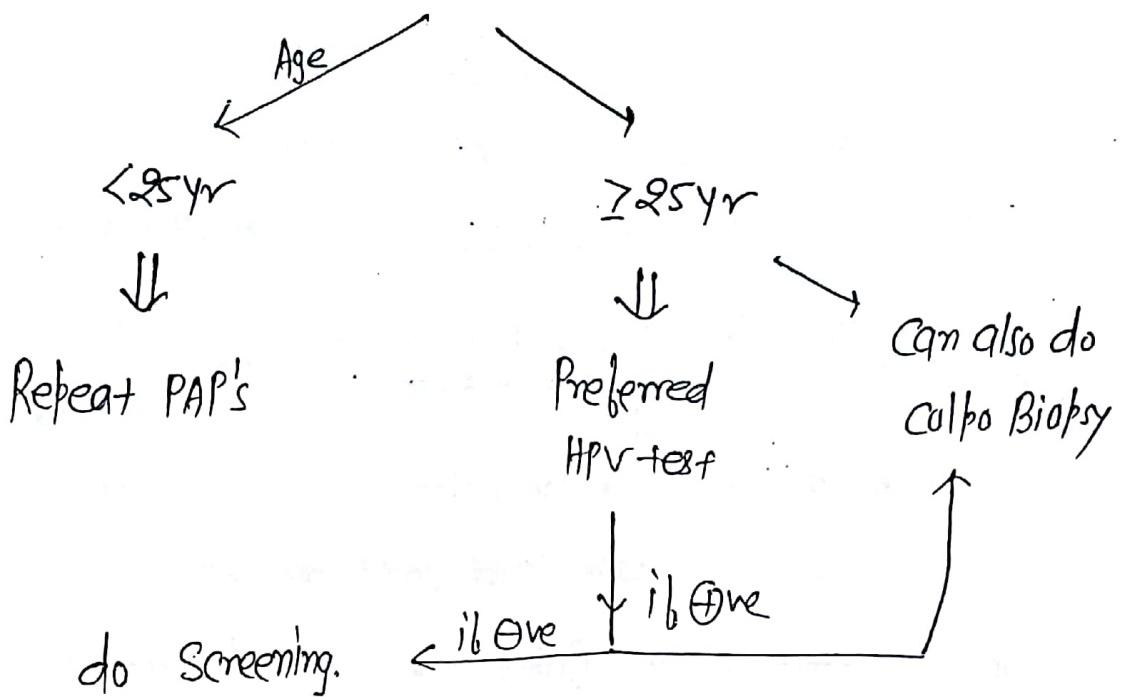
Moderate dysplastic
 \downarrow

Lower 2/3 of the
epithelium \rightarrow dysplastic

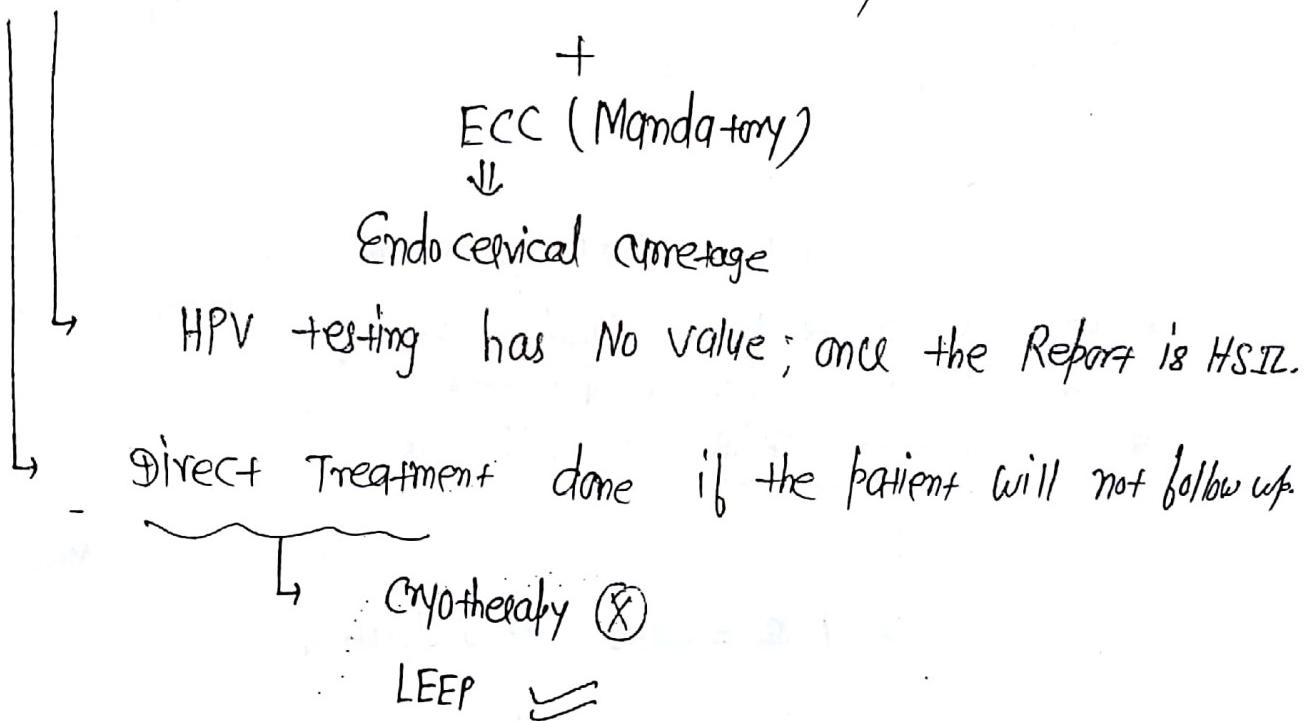
Severe dysplastic
 \downarrow

? Lower 2/3rd of
the epithelium - dysplastic

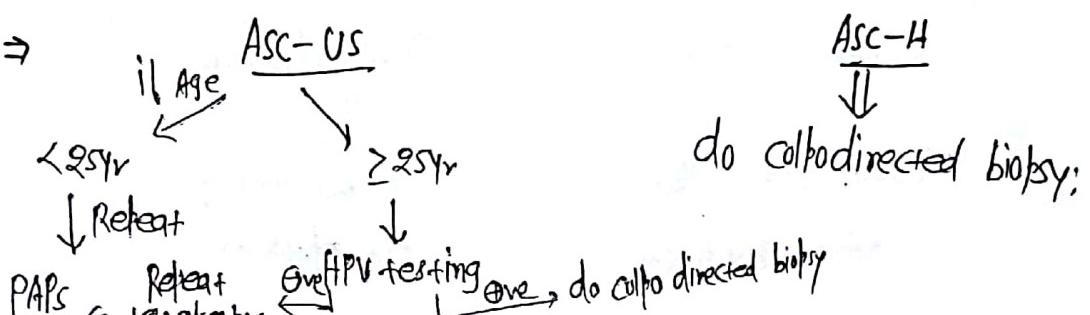
LSIL \Rightarrow if the case is of LSIL



HSIL \Rightarrow do colpo directed Biopsy



ASCUS \Rightarrow



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* If colpo Reports — confirming the Lesion

CIN-1 → Ca 1-1.



Follow up



Co-test yearly for 2 years



↳ Usual time of
CIN1 to Regress

If Persistent for 2 years



Treat

CIN-2 → 5-10 (cancer)] Treat.
CIN-3 → 12-40-1.]

* How to Treat ⇒

Cryotherapy > LEEP > Conization

Who is eligible — 21 criteria

- i) Entire Squamo-columnar Junction visible
 - ii) Entire lesion should be on Ectocervix;
 - iii) if lesion should occupy < 75% of Ectocervix
- all are known by visual inspection by acetic acid

do CRYO ABLATION → OPD Procedure

Apply cold gases (CO_2 / Nitrous oxide)

↳ destroy the cells in tissue

Mechanism of cryoablation

↳ Freeze - Thaw → Freeze

do crystallization of Intracellular water



Causes desiccation

Pain Managed by Analgesics

Long term complication → Persistent watery discharge

↳ bleeding is Not a complication of cryoablation.

- * Laser Ablation do → i) Depth of Lesion is More,
→ ii) Lesion extends on the vaginal wall

* LEEP = LLLETZ →

↓ ↓

Loop electro- Large loop
Surgical excisi- excision of
onal procedure Transformation zone

→ electric current
do cutting + coagulation

* Bleeding is Not the
SIE of LEEP.

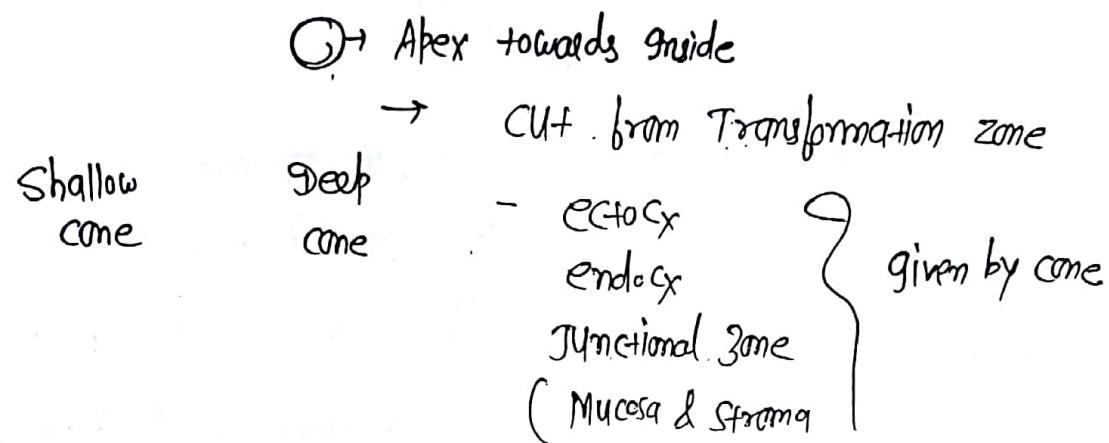
====

} No training required;
OPD Procedure!
takes < 4 min in completion of
procedure

* Cryotherapy doesn't give us any specimen; while LEEP give us
specimen after procedure. (Specimen of Transformation zone)

* CONIZATION \rightarrow Invasive;
O.T. procedure;
Remove Tissue;

* Indication of cone / C/I for LEEP \Rightarrow



- i) Unsatisfactory colposcopy
 - ↳ entire Transformation Zone is Not visible;
- ii) When there is discrepancy b/w cytology & HPE
- iii) if PAP's Smear is HSIL \rightarrow do Colpo. + ECC
 - ↳ \oplus ve
- iv) if there is Suspicion of Microinvasion
- v) if Biopsy Report Says \Rightarrow AdenoCa (Suspicion of endoc. involvement).

Q: 38 yr old Lady P₃L₃ CIN3 ??

~~a) cryo~~

- b) LEEP
- c) Conization
- d) Hysterectomy.

→ only indication to pre-invasive lesion

- ↳ i. In Recurrent CIN :
- If Patient will not follow up;
- If Suspicion of Adeno histology & family
Complete ↓
May be case of endometrial Ca;
So, do hysterectomy
- Other Pelvic Pathologies which justify a hysterectomy.

CANCER

- RIF →
- i) Early Age of 1st intercourse
 - For CX cancer
 - ii) ... 1... 1st child birth;
 - iii) Multiparity
 - iv) Multiple sexual partners
 - v) Low Socioeconomic status
 - vi) STD
 - vii) Smoking → ↑ Sq cell Ca
② AdenoCa.

viii) PreInvasive Lesion (⑦)

ix) OCP → ↑ Risk if use beyond or equal to 5yr

↳ Nullify Risk if leave for ≥ 10yr

↳ ↑ Related to Adenocarc.

x) family history ↳

Early Menarche

Late Menopause

(X) Not a R/F for Cx Cancer

Etiology of
Cx cancer ⇒

HPV

↳ also a/w cervix; vaginal; vulval cancer;
Penile; Anal; oral

High Risk HPV ⇒ Cause Cancer

Low Risk HPV ⇒ 6,11 → Cause genital warts

↓
condylomata Acuminata

↳ Laryngeal Papillomatosis

* On HPE of HPV Infected cells

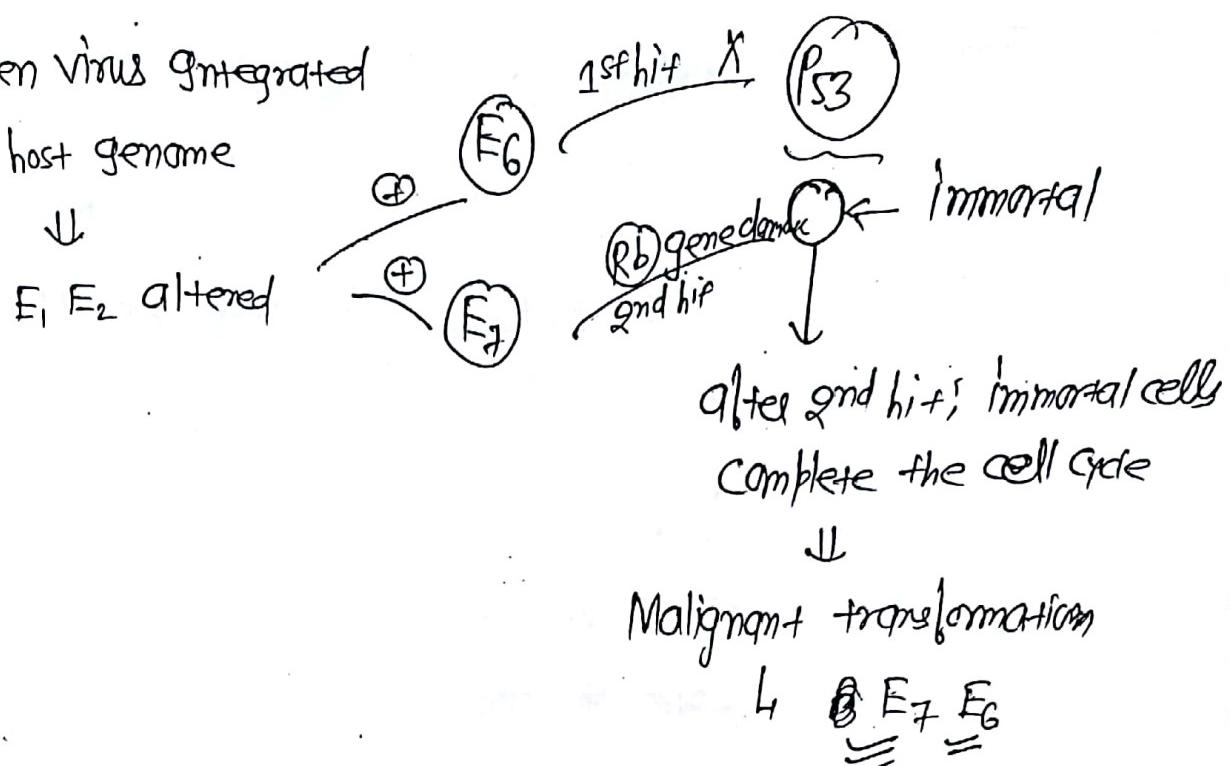
↳ Koilocytes

↳ Perinuclear halo-

Cancer ⇒ E₁ E₂ E₆ E₇ viral protein①
function

②

- * When virus integrated
in host genome



* HPV vaccines

Gardasil

Quadrivalent

- Made from Inactivated capsid protein

16, 18, 6, 11

CERVARIX

Bivalent

16, 18

- Latest FDA approved Gardasil-9 (Nonvalent)

16, 18, 6, 11, 31, 33, 45, 52, 58

- Given 0.5ml i/m

given 0.5 ml i/m

- 0, 2, 6 Month

0, 1, 6 Month

- Ideal age to give the vaccine \Rightarrow 11-12 yr
can be given \Rightarrow 9 yr - 21 yr

- Girls/Boys both taken

Girls taken

S/E of vaccine \Rightarrow Syncoal attack



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So; there has to be observation time of
15min; then send her to home

* Protection Rate - Quadrivalent Gardasil \Rightarrow 70%
Gardasil 9 \Rightarrow 95%

* M/c virus a/w Cancer Cx \Rightarrow HPV16 = Squamous
Most Specific virus a/w cancer Cx \Rightarrow HPV18 = Adeno

* For Cervical cancer \Rightarrow

M/c Age \Rightarrow 3rd-4th decade
(Shows Bimodal Peak) \nearrow 1st 3rd-4th decade
 \searrow 2nd = 5-6th decade

M/c histology \Rightarrow Squamous cell ca = 69%
Adeno ca = 25%

Large cell Non-Keratinizing squamous cell ca

M/c Route \Rightarrow Direct; Lymphatic

M/c Presentation \Rightarrow Irregular vaginal bleeding

1st presentation \Rightarrow

Most Specific presentation \Rightarrow Post coital bleeding
 \downarrow Next
 Clinical examination

clinical examination

↓ +

do PAPs Smear *

* Persistent Post coital bleeding \geq 6 Months

↓

ever with (N) PAPs

↓

do colpo Biopsy

Q 35yr P₂L₂ c Post coital bleeding ; of B 2x2 cm growth
on the Anterior lip of cervix.

do PUNCH BIOPSY.

Q M/c site of distant Metastasis \Rightarrow Lungs
In Cx cancer

M/c cause of death \Rightarrow Uremia (Renal failure)
In Cx cancer

Risk of ovarian involvement \Rightarrow < 1%.

In Cx cancer (Ovaries - Spared)

Most imp. Prognostic Marker \Rightarrow Stage > Lymph Node status
In Cx cancer

* Staging for Cervical cancer \Rightarrow

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FIGO - Clinical Staging
 \Downarrow

Investigation Should not be used to change stage of cancer \Rightarrow

USG] \times
 CT
 MRI
 PET

Not used.

Cystoscopy (to look bladder cavity);

\hookrightarrow Part of Staging

EUA (Examination Under Anesthesia);

\hookrightarrow Part of Staging

Stage 1 $A_1 \left\{ \begin{array}{l} \text{- Micro} \\ \text{- Invasion} \end{array} \right.$ - depth $< 3\text{mm}$; Horizontal limit $< 7\text{mm}$
 $A_2 \quad \quad \quad$ depth $3-5\text{mm}$ " $< 7\text{mm}$

$B_1 \left\{ \begin{array}{l} \text{- Macro} \\ \text{- Invasion} \end{array} \right.$ - depth $\leq 4\text{cm}$ } & all microscopic cond'n
 $B_2 \quad \quad \quad$ - depth $> 4\text{cm}$ " above than A_2 .

Stage 2 Upper 2/3rd of vagina

do MRI; but we can't
 \downarrow change the stage
 connective tissue

A - Without invasion of Parametrium

B - With invasion of Parametrium \hookrightarrow Clinically spreading
 Obliteration of fornices

Stage 3

A \rightarrow Lower 1/3rd of vagina is involved

B \rightarrow Spread to Lateral Pelvic wall
 (Ureter \rightarrow hydroureter)

\hookrightarrow Seen by gyn/CT \rightarrow approved to change stage
 to look for hydroureter.

Stage 4 : A → Spread to bladder and/or rectum

B → distant spread
Inguinal L.N.

* Early Stage → Who Stage 1B₁:
Cancer ↴ Primary RT → Surgery

Locally Advanced → ≥ 1B₂:
Cancer ↴ Primary RT → Chemo Radiation

Treat of Cancer Cervix ⇒

1A₁ → if Family is complete



↳ Hysterectomy

if Family is Not complete

(Type-1 Hysterectomy / simple extralaparoscopic hysterectomy)



Fertility preserving conization.
(Therapeutic conization)



→ Cut Uterus; Cx & Overlying fascia And Remove

1A₂ → if Family is complete

↳ Type 2 Hysterectomy + Pelvic LN

(Wertheim / Modified Radical) dissection

Cut Midway b/w
Uterosacral Ligament
and/or cardinal
ligament Remove Uterus;
Cx & half of Parametrium



→ Lateral Pelvic wall

→ Parametrium

IA₂ - if Family is Not complete

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do Radical Trachelectomy



Remove Gx + Parametrium + Pelvic LN

Spares Uterus

IB₁ - if Family is complete



Type-3 Hysterectomy (Meigs / Radical hysterectomy).

+

Pelvic L.N. dissection

+ When we Remove all the Lymph Node

Para-aortic LN Sampling.

↳ Few L.N. Remove

Inset on the

Lateral Pelvic wall we

cut the cardinal / uterosacral

Ligament.

Type 1, 2, 3

Hysterectomy



Only for Malignancy
(Piver Rutledge classification)

Q8. Cancer 1cm ; 4mm deep and 9mm Ig Limit II Stage \Rightarrow
 In case if tumor $\leq 2\text{cm} \Rightarrow$ type 2 Hysterectomy
 (Wertheim's)

Mic Surgery in the Cx cancer

* I.B₁ \rightarrow Family is Not complete



if $\leq 2\text{cm} \Rightarrow$ do Radical Trachelectomy.

CHEMORADIATION

~~I.B₁~~ Stage $\geq 1B_2$



Chemo-therapy + Radio-therapy
 (give concurrently)
 Radiation sensitizers

Chemo Agent \Rightarrow cis-platin > SFU



In 2A1 ($< 4\text{cm}$)

Type 3 hysterectomy

Upper 2/3rd of vagina

Removed (or half of vagina
 Removed)

Not Recommendation; but we do.

* Radiotherapy Distant \rightarrow External beam RT
 Close

(EBRT) - "Kos" Teletherapy

Brachytherapy (Intra-cavity)

Mic Wed = Iridium,

* 1stly EBRT gives then followup by Brachytherapy,
 Mic Wed = cesium

4121

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* EBRT gives to Pelvis

↳ Dose \Rightarrow 50 Gy in 5 weeks

(5 fraction every weeks)

* EBRT gives to Pelvis + Abdomen (Extended field RT)

↳ Dose \Rightarrow 30 Gy

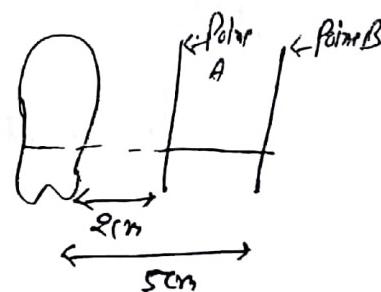
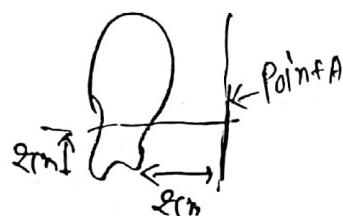
* Brachytherapy gives to Pelvis



- 2cm above external
OS

- 3cm Lateral to Point A at
the Same Level

- 2cm Lateral to cervix



- Corresponds to Paracervical
Lymph Nodes ; Ureter

- corresponds to ureter

- Dose \Rightarrow 8000 cGy

6000 cGy

Can tolerate Radiotherapy

Radiosensitive \Rightarrow Ovary > Rectum > Bladder > Vagina
 Order
 ↓ Most Radiosensitive ↓ can tolerate Radiotherapy
 (can't tolerate Radiotherapy)
 for tolerant Mobilize it.

* Adjuvant Chemoradiation

↓ Give Post-operatively

Reports says \Rightarrow i) Lymph Node \oplus
 ii) Parametrium Invasion \oplus
 iii) Tumor Margin all \oplus ive

Q.Q. What to do in Recurrence of Cx cancer \Rightarrow

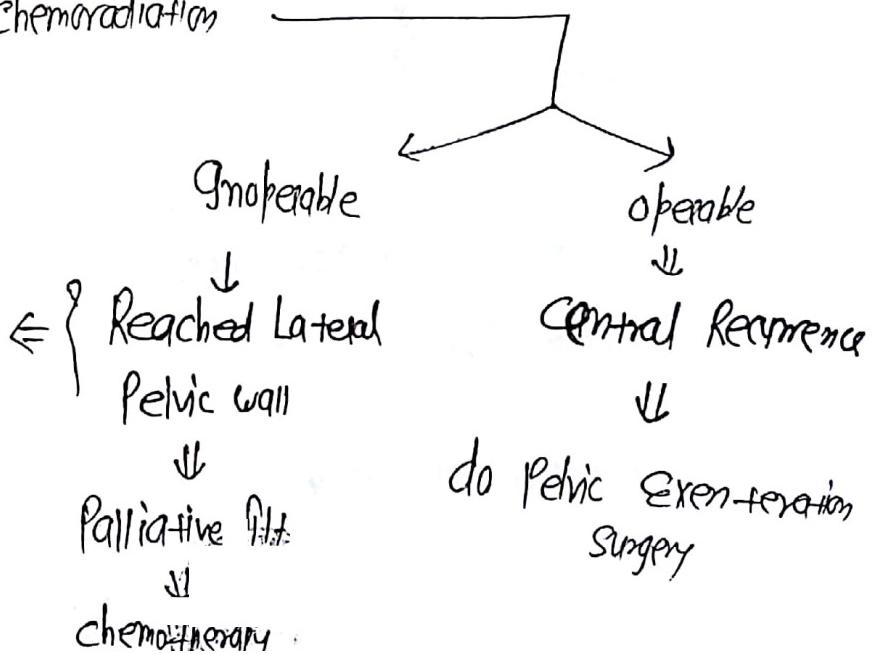
Primary fit if Sx $\xrightarrow{\text{do}}$ Chemoradiation

if Primary fit Chemoradiation

seen by Looks for
hydronephrosis;

ULV^{local} Limb edema;

Pain Radiating on one Leg.



Q8 Known case of Cancer cervix 3 cm growth upper vagina
 is involved in Parametrium; CT - hydrocoele; cystoscopy
 bulbosa edema of bladder Mucosa

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Stage 3B.

doesn't mean invasion of bladder; i.e. Lymphatics obstruction, so, Not change the stage

OVARIAN CANCER

- Not very common like of cervical ca
- No specific sign & Symptom
- Routinely Not Screening for ovarian Cancer



Pt. comes in Adnexal Mass

(More commonly in)
 benign & Malignant
 ; Not a Rule

Benign

Malignant



In Reproductive Age



In extremes of Age

{ Pubertal
 PostMenopausal

Palp +

-

V/L

B/L

Cystic consistency

Variable consistency

Benign
tender

Malignant
Non-tender

IOC for Adnexal Mass \Rightarrow TVS

{ U/L; Unilocular; Anechoic } \Rightarrow Benign cyst
Most commonly

* M/C cyst of the ovary \Rightarrow Follicular cyst

B/L; Solid component; Septae; Tes vascularity; Ascites; Enlarged LN/
(thick > 2-3 mm) / \ Matted Bowel Loop
Ground Septae
Mass



Malignant cyst. (Highly Risk of Malignancy)

Sx \Rightarrow Malignancy - Laparotomy

Benign - Laparoscopy

Indication

- i) High Risk condition on USG
- ii) Ovarian Mass $> 7\text{ cm}$
- iii) Adnexal Mass $> 10\text{ cm}$
- iv) Raised CA125 ('N value < 35)
↳ only in Post Menopausal age group.

IV Mass present as acute Abdomen d/t Rupture
d/t Torsion

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* Benign looking Mass
(UL; Unilocular; Anechoic Mass)

in Reproductive Age
group patient

↓ check size

if 3-5cm ⇒ Wait & Watch (Maybe follicular cyst)

if 5-7cm ⇒ Follow up 6 wks; 12 weeks

* Give OCPs → Pain
→ Menstrual irregularity
contraception

it prevents occurrence of New adnexal cyst;
but Nothing do currently exist cyst

in extremes of Age
patient

↓

Tumor Marker

• Pubertal (Adolescent)

↓
AFP + Hcg

- Post Menopausal

↓
CA-125

* Adnexal Mass in a pregnant women ⇒

in 1st trimester ⇒ Wait & Watch;

in 2nd trimester ⇒ i) High Risk features

↓
Safer time for
elective surgery

ii) >10cm (size)

iii) Acute Abdomen

Cyst seen common in ♀ ⇒ i) Theca Lutein cyst → Hcg
(Pregnancy Marker)
↳ also in Clomiphene citrate therapy
(Adnexal Mass)

iii) Luteoma → In Pregnancy
↓
virilizing ovarian tumor
↳ In Mother

Less common - In fetus

- Spontaneously Regress after ♀.

More the ovarian cycle
↳
More the Risk of ovarian tumor
More the estrogen
↳
More the Risk of ovarian tumor

* RIF for ovarian tumor → i) Early Menarche;

ii) Late Menopause;

iii) Infertility;

iv) Obesity;

v) BRCA 1
2

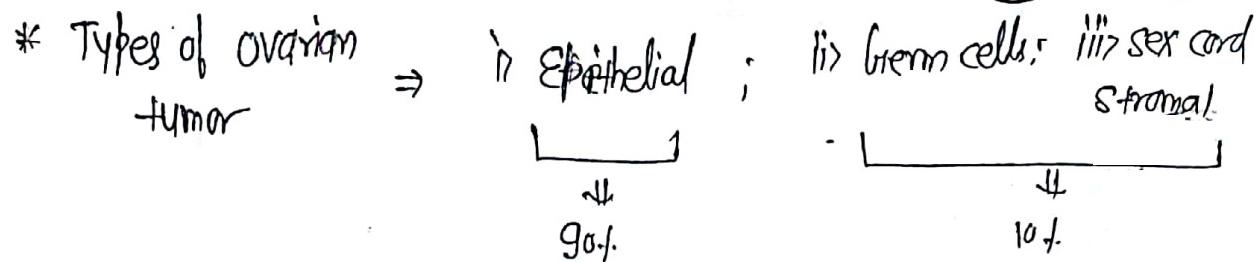
HNPPCC

vi) Smoking (only in Mucinous variety of ovarian tumor)

vii) PCOS

Protective for ovarian tumor ⇒ i) O.GP
ii) Breast feeding;
iii) Only Anovulation (Not w/o Any disease)
iv) Salpingectomy
Tubal Ligation
Hysterectomy] Ascending Mitogens
] don't Reach ovary

* Fallopian tube ca; ovarian ca & peritoneal carcinomatosis
↳ All have common origin → Fallopian tube



(A) Epithelial ovarian TUMOR (Features) -

- Serous - 75%
 - Mucinous
 - Endometrioid
 - Brenner
 - clear cells
- ? 10f each

Presentation Peak age - 60 yr.
(6th-7th decade)

- B/L
- Non-specific sign & symptom,
↳ Irritable bowel Sx
- Late stage
- High Mortality Rate
- Sporadic - 90%

So; only 10f tumor are familial.

Can tend to occur
a decade earlier
(50yr)

\Leftrightarrow Gene Involved \Rightarrow BRCA 1 $\frac{**}{**}$
BRCA 2 - 2s
HNPPC - 1s,

* If First degree Relatives affected \rightarrow Risk Tes by 3 times.

Q&A If patient is K/c/o Genetic Mutation \downarrow

Go for Prophylactically Sx

Go for BSO (Beyond 35yr Risk Tes every year)

BSO (B/L Salpingo-oophorectomy)



done @ 35yr or as soon as family complete



This surgery protects >90% from ovarian cancer
also protects from Breast cancer by 50%.

2nd Line
approach

⇒ if Patient doesn't want to go for BSO



give OCP's + Screening - High Risk



High Risk { i) Strong family Breast/ovarian ca
ii) Kloclo Genetic Mutation

Screening ⇒ TVS + CA125

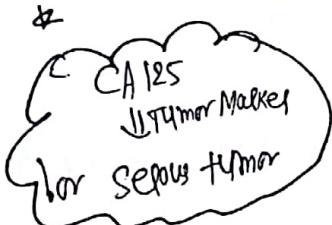
Start @ 35yr 6 Monthly / 12 Monthly

Ovarian Tumor

⇒ M/c Ovarian tumor = Serous Cyst Adenoma

M/c ovarian ca = Serous cyst Adeno carcinoma

* Mucinous Ovarian tumor ⇒ i) Decade earlier



ii) Grow to Large size (> 20 cm)

iii) Diagnosed early

iv) Better Prognosis

v) BIL (BIL in 10% cases)

vi) CEA (Tumor marker)

VII) Pseudomyxoma Peritonei

(171)

(Loculated Mucinous collections in Abdomen)

M/C Seen = tumor of Appendix; Not ovary.

* Endometrioid ovarian ca - high Risk for co-existent =
"Endometrial ca".

- ↳ 10% of total
- Epithelial
- ↳ U/L in Nature

* Clear cell ovarian ca - highest association = Endometriosis

Also a/w Endometrioid ovarian
ca.

HobNail cells + (O)
↳ HPE findings.

* Brenner - U/L; Benign; Solid lesion

↳ on HPE - Bladder Like epithelium - Transitional epithelium.

(B) GERM CELL TUMOR → 5-8% of all ovarian tumor

• Teratoma → Mature cystic Teratoma (Dermoid)

↳ Immature

• Dysgerminoma

- Embryonal cell ca
- EST (Endodermal sinus tumor) | Yolk sac tumor
- Choriocarcinoma
- Mixed tumor

M/c Germ cell tumor of ovary \Rightarrow Dermoid

M/c Germ cell cancer of ovary \Rightarrow Immature $>$ Dysgerminoma
Teratoma

* Feature \Rightarrow i) U/L (Unilateral);
of GCT which GCT has highest Risk of Bilaterality \Rightarrow

Dysgerminoma $>$ Germoid
(15%) (10%)

ii) Seen in younger girls (10-30yr)
 \downarrow

What % of ovarian tumor in this \Rightarrow 70%
Age grp. all Germ cell tumor

iii) Non-Specific signs & symptoms + (A) Precocious Puberty
(B) Acute abdomen

iv) Pick up in early stage;

v) Good Prognosis

vi) Conservatively.

b/c GCT Release hCG

\downarrow
& Submit similar to LH / FSH

EST / Yolk sac tumor

\downarrow More rapid growth of tumor

* Dysgerminoma \Rightarrow TUMOR MARKER
 \downarrow LH (HCh + PLAP)
 Fleshy; Lobulated & Tan in colour. \downarrow Not Secrete AFP.

(172)

* Endodermal Sinus Tumor \Rightarrow AFP (LH)
 \downarrow Tumor Marker
 \downarrow Not Secrete HCh.

* Choriocarcinoma \Rightarrow HCh

* Embryonal \Rightarrow HCG + AFP

* Dermoid \Rightarrow No Tumor Marker

\downarrow Rarely HCh Secrete

Rokitansky Protrubance / Tip of Goebel sign

\downarrow
 White Area Inside cyst (black) in USG.

* M/c ovarian tumor in Reproductive Age \Rightarrow Dermoid.

11 11 In ♀ \Rightarrow Dermoid

11 Cancer \Rightarrow dysgerminoma

* Dermoid \Rightarrow Benign

Risk of Malignancy (0-2-2+)
 type site

Sq cell Ca

Rokitansky
Protrubance

M/c ovarian tumor goes in torsion,

- * Germ cell cancer \subset Best prognosis \Rightarrow Dysgerminoma
 - " Worst prognosis \Rightarrow EST / Yolk sac tumor
 - * Only ovarian tumor \subset is radiosensitive
- \downarrow
- Dysgerminoma (Moderately radiosensitive)

⑥ Sex cord stromal tumor (3+)

- Granulosa cell tumor
- Sertoli-Leydig cell tumor
- Leydig cell tumor
- Thecoma
- Fibroma (Stromal tumor of ovary)

Features \Rightarrow , U/L : Occur in all age groups / Peak incidence in Perimenopausal women;

- Non specific signs/symptoms,
- AUB \longrightarrow Estrogen
or
virilization \longrightarrow Testosterone
- Early stage pick up
- don't show any Lymph Node Metastasis.
- Best prognosis;
- Granulosa cell tumor — Gnhibin
 \downarrow Secrete estrogen \rightarrow Risk of endometrial ca p.

- On HPE \Rightarrow
 - Call excret bodies (follicle) (173)
 - Rokitansky protuberance (also in dermoid)
 - Walthard cell Nest
 - Colee-bean cells (also seen in Brenner tumor)
 - Reinke's crystal \rightarrow Leydig cell tumor
 - Schiller-duval bodies \rightarrow EST tumor
 - Pseudomma bodies - serous cystadenoma of ovary

Krukenberg's tumor \Rightarrow Not a Primary tumor; Secondary tumor.

- Mic Primary tumor \Rightarrow Gastric cancer
- Spreads via Retrograde Lymphatics
- B/L in 80% cases

Shape of the ovary Maintained;
Capsule of the ovary intact

On HPE \Rightarrow Signet Ring cells*

STAGING OF OVARIAN CANCER \downarrow by FIGO; Surgical Staging.

| | | |
|---------|----------------|---|
| Stage 1 | A | \rightarrow U/L ovary |
| | B | \rightarrow B/L ovary |
| | C ₁ | \rightarrow Intra operative capsule Rupture |
| | C ₂ | \rightarrow Pre operative capsule Rupture |
| | C ₃ | \rightarrow Malignant Ascites |
| | | |

| | | |
|---------|---|--|
| Stage 2 | A | \rightarrow Cancer Spread to uterus/fallopian tube |
| | B | \rightarrow Pelvic Symmetes |

Stage 3

A → 1 ⇒ The Retroperitoneal Lymph Node

2 ⇒ Microscopic extraperitoneal spread

B → Macroscopic $\leq 2\text{cm}$ extra pelvic peritoneal spread

C → Macroscopic $> 2\text{cm}$ extraperitoneal spread

↳ If there is involvement of capsule of liver/spleen

Stage 4

A → Malignant pleural effusion

B → Paenchyma of the abdominal organ
distant spread

Inguinal Lymph Node

Stage 1/2 → early Stage

Ca

Stage 3/4 ⇒ Advanced stage
Ca

* For early diagnosis
of ovarian Ca ⇒ TVS

or

K/c/o ovarian Ca ⇒ Best ⇒ CT-scan
↳ Can't do staging CT-scan

* On follow up: If CA 125 ↑
↳ It means Recurrence happen
do PET scan

Staging Laparotomy \Rightarrow Steps

- i) Midline Vertical Incision
↓
Never give Pfannenstiel Incision or transverse Incision; Rarely ParaMedian Incision given
- ii) Ascites → Sample taken → Cytology for Malignant cells
OR
Saline wash → 50-100ml → Cytology for Malignant cells
- iii) Inspection & Palpation of all abdominal organs,
- iv) Random Peritoneal biopsies
↓
Paracolic gutters
Pouch of douglas
Surface of diaphragm → Scrapping (acceptable)
- v) TAH \equiv BSO (Pan hysterectomy)
↓
Type 1 hysterectomy
- vi) Gastrocolic Omentectomy.

vii) Pelvic & Para-aortic LN Sampling

viii) closure

* In Stage 3/4 \Rightarrow Primary RT \Rightarrow Surgery
 \Downarrow
Debulking sx

* Post-operative \Rightarrow Chemotherapy.

* Conservative sx for ovarian cancer \Rightarrow V/L Salphingo-oophorectomy
 \Downarrow eg \Rightarrow • Germ cell tumor
(V/L; younger age patient)

- Stage IA (if fertility is desired).
- Borderline epithelial ovarian tumor
 - epithelial ovarian potential = low Malignant potential
(Stromal Invasion - Absent)
 - Very good prognosis
 - V/L
 - decade earlier

Post-operative chemotherapy

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- 4 Epithelial \Rightarrow all stages except \Rightarrow 1A & 1B graded
cell ca Need Post-op. chemotherapy,

Chemotherapeutic Agent \Rightarrow Carboplatin + Paclitaxel { 6 cyc.
 ↓
 Cisplatin + Paclitaxel }
 given i/v + Peritoneal.

Germ cell ca
Sex cord ca \Rightarrow All Stages Need chemo. except \Rightarrow
Dysgerminoma stages II
 No Need of chemo.

Only Advanced stages Need chemotherapy.

Chemo. Agent \Rightarrow BEP (Bleomycin; Etoposide; cisplatin),

ENDOMETRIAL CANCER

Etiopathogenesis \Rightarrow ↑ Estrogen (unopposed)
 ↑ Menstrual cycles

RIF \Rightarrow Early Menarche;

Late Menopause;
PCOS;

IV Infertility
V Obesity
VI HTN
VII Diabetes

VIII Tamoxifen → highest - 70%

IX BRCA1, 2, EINPC → Lynch syndrome

↳ also ovarian ca; Not of Breast
cancer

X HRT (only Estrogen)

Protective
of Endometrial ca

Smoking
, OCP (↓ Risk by 60%)
(↓ Risk of ovarian ca by 50%)

Exercise
Green tea

Histology ⇒ Type 1

Endometrioid

Most common in 80%
cases

Estrogen responsive

Pregnant

Good prognosis

Type 2

Papillary; serous; clear cell

In 20% cases

Non responsive

⊗

Poor prognosis (worst prognosis)
clear cell

Type I
Gene association \Rightarrow PTEN \leftarrow Gatakeber Gene
KRAS

Microsatellite deletions

p53

High grade serous show p53 Mutation

Low grade serous show KRAS Mutation

Pri Menopause

Later More Tage More type II

Early Menopause

Obese Lady

thin Lady,

* Pre Invasive Lesion \Rightarrow
which changes into the
endometrial ca

Endometrial hyperplasia

on HPE by taking endometrial Biopsy.

Kla "cystic glandular hyperplasia": }
Glands & stroma both proliferate

Simple hyperplasia

Cloac Atypia \rightarrow 1+

Complex II

" \rightarrow 3+

Simple \subset Atypia

\rightarrow 8+

Complex \subset Atypia

\rightarrow 29+

Glandular Proliferation is Much more than stromal proliferation

Back to Back arrangement of gland.

Hyperplasia C/ut Atypia

↳ Given Progesterone 1stly

M/c \Rightarrow MPA \times 6 Months

(daily) \downarrow

Repeat Biopsy

also use DMPA

Mirena

Hyperplasia C Atypia

Reconfirm \leftarrow do Hysterectomy (TOC)

(Frozen section)

↳ Intraoperatively send & get a
immediate report & reconfirm,
 \downarrow if not possible

do Endometrial Sampling (Optional)

(FC + Hysteroscopy)

↳ Functional Curettage

2nd Line

\Rightarrow if wants Preserving the Uterus

Reconfirm on Repeat Endometrial Sampling

(FC + hysteroscopy)

2ndly Progesterone (Prefer - Megestrol Acetate & 6 Months)
Other MPA
Mirena

(177)

* Simple hyperplasia
Cystic glandular hyperplasia

→ Metropathia
Hemorrhagica

Age → 40-45 yr

8 weeks of Amenorrhea → History of bleeding

(Anovulatory cycles
b/c Unopposed Estrogen)

↓
Painless

Endometrium → Absence of Secretory Pattern
On HPE

Ovary → Swiss cheese appearance

Ca → cyst

TOC → 1y.

TOC ⇒ Progesterone

* Peak age for endometrial cancer ⇒ 60 yr (5-7th decade)

M/c presentation / 1st presentation = Irregular vaginal bleeding

Most specific presentation = Post Menopausal bleeding (PMB)

% of PMB have endometrial Ca = 10%

M/c cause ⇒ Senile endometrial Atrophy
(Atrophic endometrium)

M/c cause of PMB in India → Ca cervix

* M/c Cause of Pyometra \Rightarrow Endometrial ca
in India \Rightarrow cervical ca

* M/c Route for endometrial ca \Rightarrow Direct spread
II " ovarian ca \Rightarrow Tumor Exfoliation

* No Routine Screening done for endometrial ca

↓
if pt. is K/C/O " HNPCC" - do Routine Screening

↳ age 35yr

do functional curettage (FC)

6 monthly / 12 monthly.

* Any women comes \in Post Menopausal bleeding

↳ Rule out Endometrial ca

* if women ≥ 45 yr \subseteq AUB

↳ Rule out Endometrial ca

Next Step

do TVS (see endometrial thickness)

Q.C. to AcoS if thickness $> 4\text{ mm}$ $\xrightarrow{\text{high Risk for}}$ high Risk for

AIIMS May 18 II American Radiological $\geq 5\text{ mm}$ Cancer

if endometrial thickness $< 4\text{ mm}$ / $< 5\text{ mm}$
 (ACOG / American Radiology)
 L No Yes In Risk of Cancer

(178)

* IOD \Rightarrow

Endometrial Biopsy

* TVs do
Polyp

to Rule out other pathologies like fibroid,

O.P.D Procedure ; Aspiration do
(Endometrial Aspiration cytology)

↓
we use "KARMAN'S CANNULA"

Other device "PIPELINE DEVICE"

"VATIRA ASPIRATOR".

* Gold Standard
technique to Rule out
endometrial ca

\Rightarrow Functional Curettage + Hysteroscopy

May (D&C)

use synonymous to Functional Curettage
O.T. + Anesthesia Needed

↓
Most invasive

\Rightarrow 1stly do cervical curettage - (Endocervical
curettage)

↓ then

dilate internal os

↓ then

Endometrial curettage

if 1stly do endometrial
cells comes in cervix.

Condition When Functional curettage done

- ↳ i) Endometrial biopsy - Benign ; while Symptom Persistent
- ii) Endometrial biopsy - No Endometrial cells seen
- iii) Hyperplasia \subset Atypia
(preserve the Uterus)
- iv) Cervix Stenosed

* Should we do PAP Smear in Post Menopausal bleeding
Yes.

Staging \rightarrow FIGO \rightarrow Surgical Staging

Stage 1 { A - only Endometrium or < 50% of Myometrium
Intraoperatively { B - > 50% of Myometrium
Cut open the
Uterus

Stage 2 - cervical spread

\Downarrow
only if cervical stroma is involved

Stage 3 A \rightarrow Cancer Spread to Serosa / Adnexa
B \rightarrow " Vagina / Parametrium
C₁ \rightarrow \oplus ve Pelvic LN
C₂ \rightarrow \oplus ve Para-aortic LN

Stage A \Rightarrow Spread to Bladder and/or Rectum (179)
 B \Rightarrow Distant spread / Regional LN

Staging Laparotomy

\hookrightarrow Omentectomy is Not done Routinely

\hookrightarrow for type 2 Endometrial Ca.

do TAH $\bar{\in}$ BSO

\hookrightarrow type 1 — Stage 2 — Type 3 hysterectomy
 \downarrow
 Cervical spread

Pelvic & Para-aortic \Rightarrow Type 2 cancers
 LN Sampling \Rightarrow L.N dissection

Stage 3/4 \Rightarrow Advanced stage Ca

\hookrightarrow Primary Tx \Rightarrow Debulking surgery

Post-operatively

Low Risk

- all three
 - a) Endometrioid
 - b) Grade 1
 - c) only endometrium

Intermediate Risk

In b/w Low & high risk

High Risk

- a) Stage 3/4 disease OR
- b) Type 2 cancer

Risk of Low Risk \Rightarrow Need No Post-operative RT; do Follow up

Risk of Intermediate Risk \rightarrow Pelvic Radiotherapy

Risk of High Risk \rightarrow Chemotherapy + Radiotherapy
 \downarrow
(Cisplatin + Paclitaxel)

PROLAPSE $\xrightarrow{\text{Recent classification}}$
 \downarrow POP-Q classification
 \downarrow Pelvic organ prolapse quantification
 \downarrow Reference \rightarrow Hymen.

* Delancey Level of Support

Level I - Uterosacral Ligament + Cardinal Ligament
 \downarrow weak $\xrightarrow{\text{causes}}$ Uterocervical descent

[earlier classified as

1st degree \rightarrow descent above vaginal opening

2nd degree \rightarrow Uterus descent at the level of vaginal opening

3rd degree \rightarrow Uterus descent outside the vagina

Proxidencia \rightarrow Uterus completely outside the vagina
 \downarrow Fundus is also outside]

Apical Prolapse

Enterocele \rightarrow Prolapse of GUT into pouch of Douglas.
Vault Prolapse (after hysterectomy)
 \downarrow

Uterocervical descent

Level 2 \Rightarrow Paravaginal tissue & their attachment
 fascia covering Levator Ani 180

Weak $\xrightarrow{\text{causes}}$ Cystocele (Upper 2/3rd Anterior wall)
 M/c type of Prolapse

Level 3 \Rightarrow Perineal body & Muscle attach to it

Weakness $\xrightarrow{\text{causes}}$ Rectocele (Post. vaginal wall) $B \Rightarrow$ BULBOSPONGE SW;
 $L \Rightarrow$ LEVATOR ANI MUSCLE;
 $E \Rightarrow$ EXTERNAL ANAL SPHINCTER

* Prolapse is disease of older age.

- RIF of Prolapse \Rightarrow i) No. of deliveries & process deliveries
 (Prolonged Labour) $D \Rightarrow$ DEEP TRANSVERSE PERIN. MUSCLE

Instrument delivery

Episiotomy "

Perineal tear

obstructed labour

ii) Tel Age (d/t loss of estrogen)

* Younger age women also have Prolapse of uterus by →
 May

i) Connective tissue d/o.

ii) Congenital elongation of cx

iii) Spinal cord injury

- * Smoking increases Risk of Prolapse
 - * Intra-abdominal Pressure ↑ ⇒ ↑ Risk of Prolapse

$\underbrace{\hspace{10em}}$

↳ Chronic cough; Ascites.

Case I → Old woman P₃L₃; 3rd degree Uterocervical descent
- + cystocele + Rectocele

Uterocevical descent → do Vaginal hysterectomy
↓

Sequence of cutting
& clumping the
structure in TAD

Sequence of cutting & clamping of Sequence

Reverse the sequence
of vaginal hysterectomy

Cardinal ↓ Ligament

Cefalme Artery

Uterus ovary Pedicle

* Sequence of cutting & clumping the structure in TAH C BCO \Rightarrow

* Usually we prefer vaginal hysterectomy; but it is difficult in — Uterine size $> 12\text{cm}$ (181)

Obese

Pelvic Adhesions.

* Urinary Tract Injuries

↳ Laparoscopic Sx \gg Abdominal \gg Vaginal
hysterectomy \gg Vaginal hysterectomy.

↳ highest Risk for Urinary tract injuries \downarrow in Benign cond'

↳ Seen in hysterectomy for Malignancies
(Wertheim's hysterectomy)

* Rx of cystocele \rightarrow Anterior colporrhaphy
(vagina)



Interrupted Suture

Rx of Rectocele

\rightarrow Posterior colpotomy

↓
Strengthen the Levator Ani Muscle

VH + Ant. colpo

+ Posterior colpopexy or rectocele



VH + Pelvic Floor Repair

+ Enterocoele Sx



Ward Mayo Sx **

do prophylaxis; also do
for vault prolapse

McCall's culdoplasty

vaginal repair

Other surgeries for Enterocoele \Rightarrow Moscheowitz Repair

Both are Abdominal Repair:

Not used nowadays

Halligans Repair

Support the vaginal vault

Uterosacral Ligament in McCall's culdoplasty

2nd case

Younger - Reproductive + Prolapse

Age group

(Uterocecal descent).

can't do hysterectomy

TOC \Rightarrow

Sling's Surgery

Modified Shridhers abdominal surgeries

Anterior sling

(If we tie at anterior aspect of sacrum)

Posterior sling

(If we tie at posterior aspect of sacrum)

* Pyramidal Sx

Autologous (femoral fascia lata)

- and - Omentum or external oblique muscle

* Khanlals Sling.

Khomma's sling

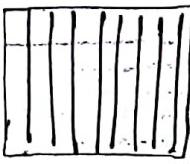
(182)

↳ one end at Posterior aspect of Gastros;
Other end Anterior Superior Iliac spine.

Shridhar sling → Posterior sling

↳ Melselene tape used
On the left hand side first pass through
Psoas Muscle (Form Psoas hook) then goes to Sacrum
 ↓
to prevent compression & obstruction
of sigmoid colon / Rectum.

VikRud's sling → Composite sling (Anterior + Posterior sling)

Abdomino cervicopexy ⇒ For putting Mesh we have to
clear Pre-Sacral Area; technically
difficult; but Results are best.
 Mesh

 ↓
 higher complication

* Non Sling surgery ⇒ Manchester Repair

↳ * done in Reproductive age women who completed child bearing.
 (Klas "Fothergills Urogen")

- also done in Congenital Cx Elongation
- Process \Rightarrow UCL (Uterocervical Length) estimation
- D&C (\rightarrow prevent the complication of cervical stenosis)
- of Manchester Repair
- Cervical Amputation
- Reattach Cardinal Ligament anteriorly
- Cover the Cervix \rightarrow C vaginal Mucosa
- Cystocele done
- Rectocele Repair done

if the UCL less ; don't do sling operation ; do
 Manchester Repair UCL = Uterocervical Length

(N) UCL Length \Rightarrow 6 to 8 cm.

* Shirodkar Modification of Manchester

- ↳ Can be done even if woman want to conceive.
- ↳ All steps all same in it except there is No cervical Amputation in it.

3rd case

old woman + co-morbidities

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PAC
 (Pre-Anesthetic
 Check-up)

— fit for short sx.

Latzko colpocleisis
 ↓

Rx → LeFort's colpocleisis done for Vagina fistula.

Denude Mucosa - Anterior vaginal wall

Denude Mucosa - Posterior vaginal wall

Meas. Scraps
 Mucosa from Anterior & Posterior vaginal wall
 1stly we Rule out Cx Cancer & Endometrial Cancer

& Patient can't be sexually active

- Done under Local Anesthesia, { colpocleisis → closure of vagina }

4th case

Co-Morbidities + old woman

PAC → Unfit for short sx.

Rx → Pessary → Space occupying device
 inserted into vagina

Gellhorn
 Pessary



Doughnut
 Pessary



- * Early Ovarian Prolapse - also indication of Pessary
 - Remove - 16-18 weeks.

- * Other indication of Pessary - Fuerferium

* Ulcer on Prolapse part

Relative
cl/I for
Persony

Decubitus Ulcer

Seen d/t Venous congestion

↑ t ⇒ Reposit

How to Reposit ??

↓ Like pads; used to absorb Menstrual Flow

Use Tampon (Hygroscopic) Antiseptic in Nature

↓ Use Glycerine Acridine (Tampon Soaked in it)

also use Local Estrogen Creams for faster healings.

healing Requires
blc surgeon Never
Operate on Nuded
Oneq.

5th case

Women who have Vault Prolapse

Abdominal Surgeries

- Abd. Sacrocolpopexy (Best) Technically difficult

- Uterosacral Suspension

↳ Mic done

Vaginal Surgeries

- Sacrosphincter fixation

- Uterosacral Suspension

Suture the vault to sacrosphincter ligament

Leborre

Colpocleisis can also done in vault prolapse (184)

In Short time Sx.

365

* All all Sx of Vault Prolapse except colposuspension:

Burch colposuspension

Gold Standard Surgery
for SUI

TOC for colposuspension \Rightarrow

done in Stress Urinary

Incontinence (SUI)

MMK colposuspension (Marshall -
Marchetti - Kranz surgery)

Not done
Support the urethra by Periosteum
of Symphysis Pubis

TVT / TOT

Intravaginal

tape

Trans obturator tape

Mid Urethral Sling

Kind of Abdominal Surgery

- Support the proximal urethra by Cooper's Ligament.

They all as good as Burch colposuspension

i) Day-care Sx;

ii) Lesser complication;

iii) Vaginal surgeries;

* Trans vaginal tape \Rightarrow Reparative Surgery

Trans obturator tape \Rightarrow Non Reparative Surgery

In it we enter the Space of Retzius

So; More complication than TOT, so;
we prefer TOT Now a days.

* Pt C Vault prolapse + SUI??

\Downarrow
Abdominal Sacrocolpopexy

Rx \Rightarrow Burch colposuspension.

* 1st Line Rx of SUI \Rightarrow Kegel's exercise i.e. Pelvic floor exercise

* Drug Rx of SUI \Rightarrow DULOXETINE (only drug Rx of SUI).

FIBROID

- Smooth Muscle tumor of Uterus $\frac{Q}{Q}$

- M/c benign pelvic tumor in females. $\frac{Q}{Q}$

- Act Estrogen; So; Seen in Reproductive Age

In > 35yr women; It is More Common.

- In PostMenopausal women \rightarrow Regression seen.

- In Pregnancy — Most of the fibroid don't enlarge.

Types →

grow within Myometrial wall,
k* \rightarrow IntraMural (M/c) \Rightarrow 75%

(185)

Subserosal

↳ Most fibroids to begin w/

(grow outward toward
the peritoneal surface)

Sub Mucosal

Intramural & Layer converted

↳ grow toward into different types
the uterine cavity,

Hysteroscopic appearance (Womstekers classification).

↳ Type 0 \rightarrow Completely Intra cavity

Type 1 \rightarrow $>50\%$ Intra cavity i.e. $<50\%$ in Myometrium

Type 2 \rightarrow $<50\%$ Intra cavity i.e. $>50\%$ in Myometrium

↳ can't be removed hysteroscopically

M/c presentation \Rightarrow Asymptomatic

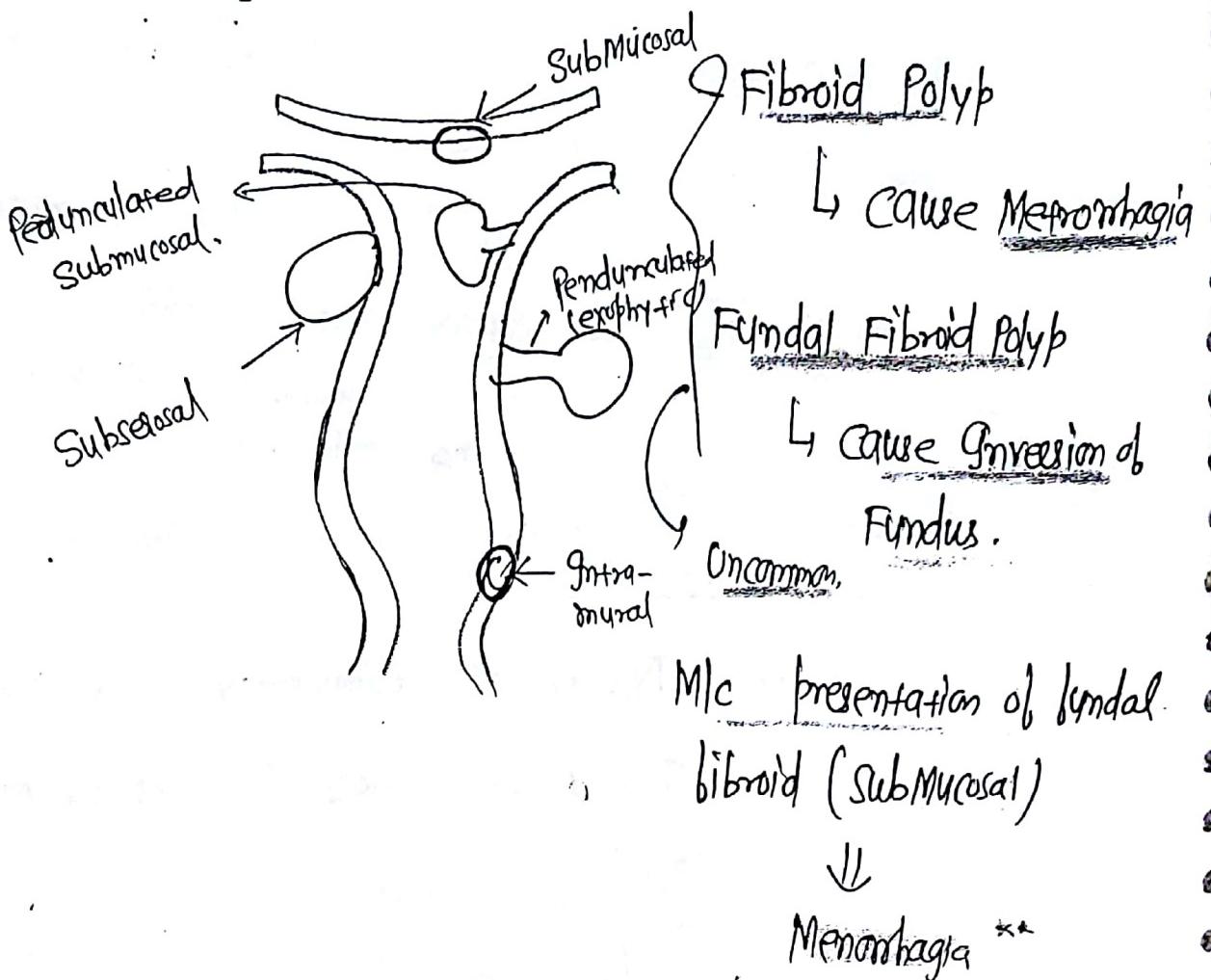
M/c symptom \Rightarrow ① Bleeding \rightarrow Menorrhagia

Cycles — Regular
Flow — ↑↑↑

M/c causing which fibroid

SubMucosal

while Subserosal fibroid Not giving bleeding



② Pain → if it undergoes - degenerations

Torsion - Pedunculated Subserosa

M/c degeneration → Hyaline **

Least common " → Sarcomatous **

Rare Malignant change

↳ < 0.5%

↳ PostMenopausal fibroid enlarge & pain.

* Womb Stone - Subserosal fibroid stone; (186)

↳ Old bladder stone ↓
It is eccentric calcification
Differential diagnosis

* Red degeneration → Seen in ♀

- ↓
- Stained salmon pink or red
- Fishy odor.
- Histologically: evidence of thrombosis in some vessels.
- M/c in 2nd Trimester.
- presenting as acute abdomen (Pain + Nausea; vomiting & fever)

- Mx - Conservative

N.P.O + iv fluids + analgesic

→ aseptic cond'n ⇒ No Role of Antibiotic **

↳ Fever is only reactionary

* In Fibroid → dysmenorrhoea ♂; but Not a primary complaint.

* Fibroid doesn't cause dyspareunia

Fibroid May show pressure symptom

↳ Ant wall fibroid ⇒ Urinary symptom
Post wall fibroid ⇒ Bowel symptom **

- * Ant. wall fibroid cause \Rightarrow ↑ Frequency of Migration
Post. " " " \Rightarrow Urinary Retention

*

BROAD LIGAMENT FIBROID

False
 \downarrow

Subserosal Uter. fibroid
Outgrown into the
Layers of Broad Ligament

True
 \downarrow

denovo b/w the Layer
of broad Ligament

Lateral to the Fibroid \leftarrow Uterus \rightarrow Medial to the Fibroid.

* Fibroid also prf to Inertility
Recurrent ♀ Loss }
} SubMucosal Fibroid
only **

* Pregnancy complication of Fibroid

Apart from RPL & Red degeneration

} Abruption
Pre-term Labour
Malpresentation
Dysfunctional Labour
PPH

IOC \Rightarrow USG (Hypoechoic)

↳ Well-circumscribed Masses;
Pseudocapsule

Small SubMucosal fibroid May Miss in USG



Best Ix \Rightarrow Hysteroscopy

2nd Best \Rightarrow SIS (Saline Infusion Sonography)

* Don't do MRI \Rightarrow Routinely



do in pre-op. condⁿ to know

No.

size

Location

* Old

Fibroid

- Smooth Muscle
- humor
- > 35 yr age
- Menorrhagia
- Non tender
- Irregular growth
(Lumpy Bumpy growth)*
- Grows upto uterus size of
20 week

Adenomyosis

Endometrial glands & stroma
Inside Myometrium

40-45yr age

Menorrhagia + dysmenorrhea

Tender (Half the size)

Symmetrical Growth
(Globular)*

10-12 week

Fibroid

Gx ⇒



Adenomyosis



On USG we see ⇒

Salt + pepper appearance;

Venetian blind //

Myometrial cyst

Subendo metrial cyst

Poorly defined functional zone

alternate
dark & light
band

* Degenerations / Secondary changes in Fibroid

→ Mnemonic

- 4 Avoid → Atrophy; don't tell about where endometrium starts & myometrium ends
- Red → Red degeneration;
- Hof → Hyaline degeneration, On MRI ⇒ Functional zone thickness
- (Mlc)
- Fatty → Fatty degeneration or calcification, > 12 mm ⇒ Likely adenomyosis
- Meat of chicken → Myxomatous degeneration
- Cystic degeneration.

< 8 mm ⇒ Unlikely //

For confirming ⇒ (HPE) - Postoperatively

Adenomyosis

See depth of these

endometrial glands in myometrium

II

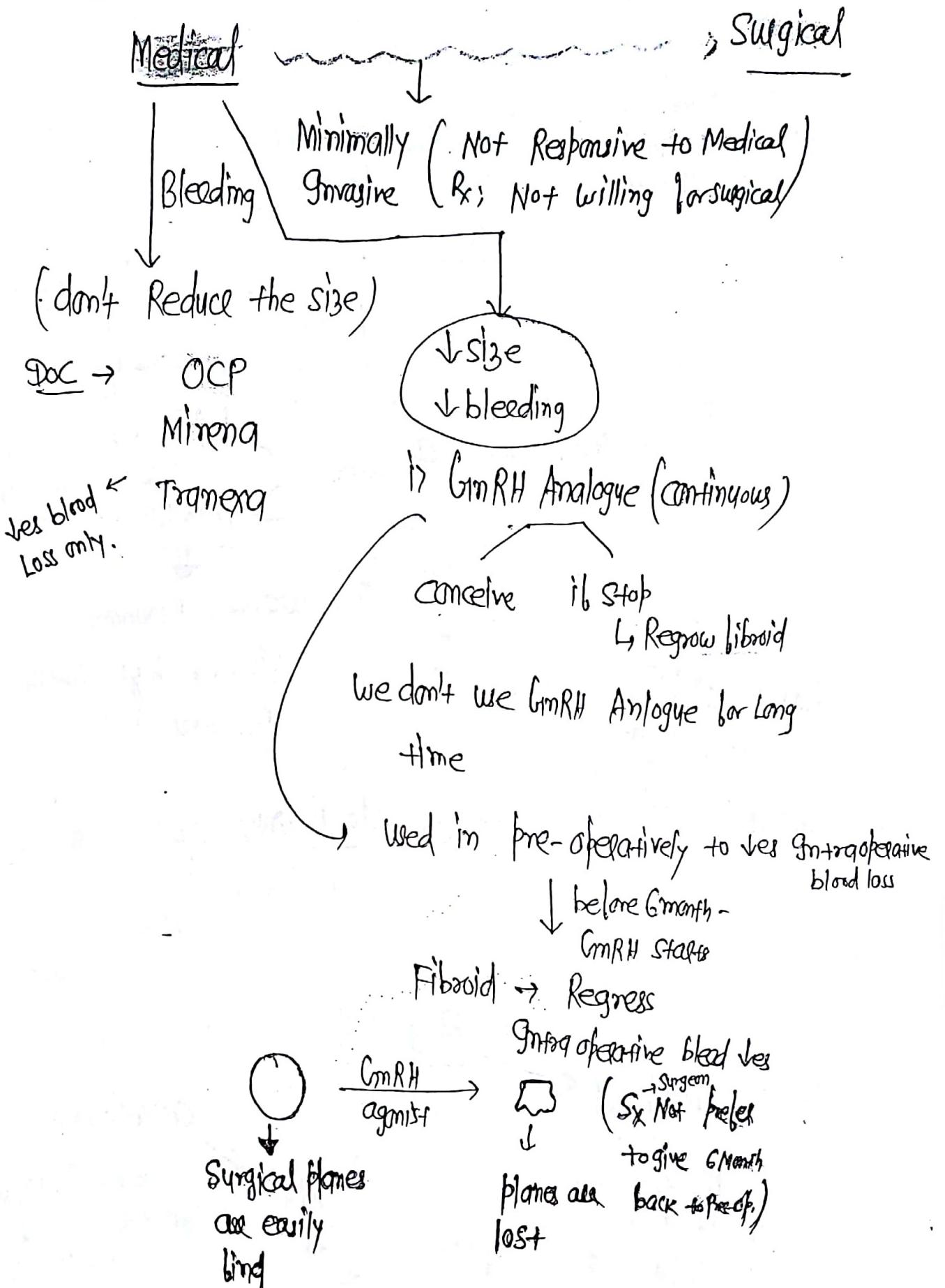
At least 1 HPF deep of 2.5 mm deep to the functional zone

TOC ⇒

Hysterectomy *

Treatment for Fibroid \Rightarrow

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Drugs that less size of Fibroid

U → Ulipristal

Are → Aromatase Inhibitor

Gynae → GnRH Agonist/Antagonist

M → Mifepristone

S → Danazol.

ii) GnRH Antagonist

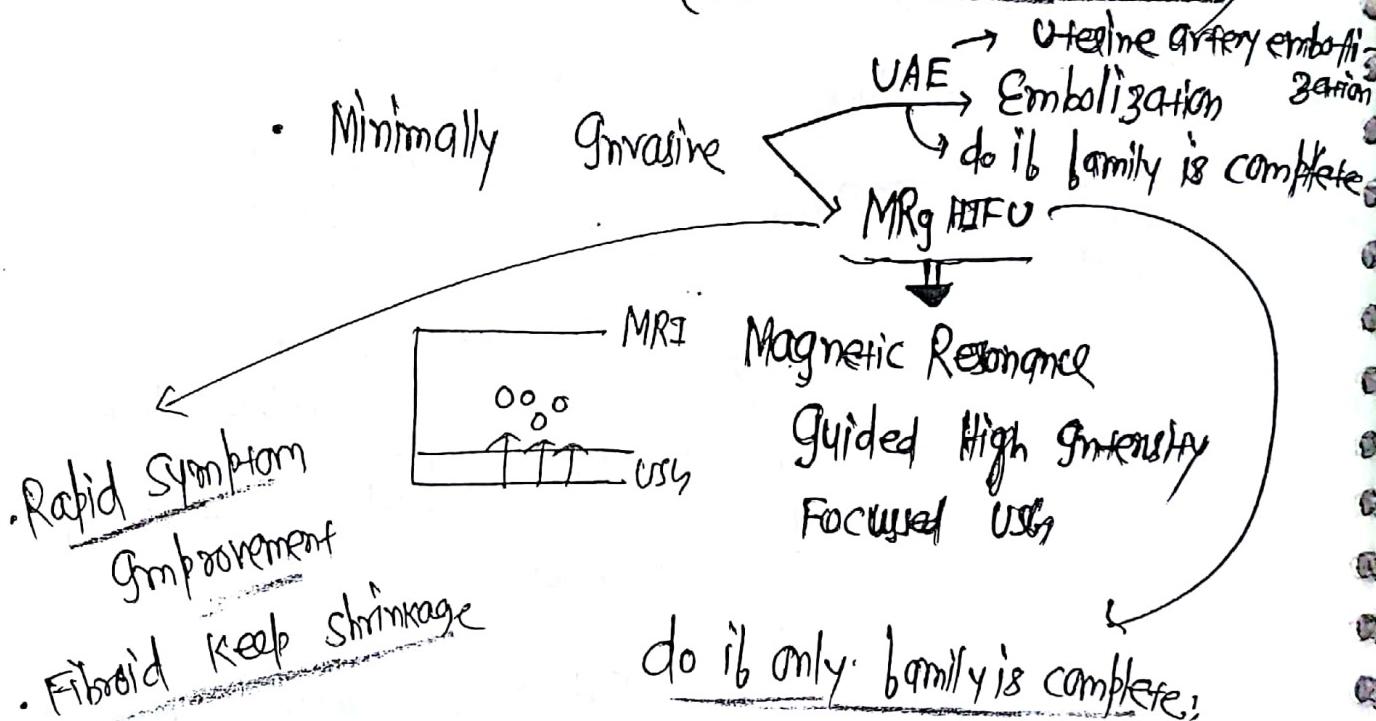
iii) Mifepristone } Progesterone (R)

iv) Ulipristal } Modulator

(Want to conceive
↳ don't give)

v) Aromatase Inhibitor - Anastrozole

(S/E - Severe Hypoestrogenic)



* Surgical P/I

Radical

Hysterectomy

Indication for TAH. → Fibroid

TAH. → Fibroid

Conservative

Myolysis

Cryo
Laser

Submucosal
(Alike or R)

Myomectomy

Hysteroscopic

Laparoscopic { others

* In Laparoscopic hysterectomy

(189)

↳ Slightly higher Recurrence Rate

↳ Not Statistically significant.

* Laparoscopy is better than Abdominal in all except \Rightarrow Recurrence

* M/c Side effect of Hyster. Scopy \Rightarrow Uterine Perforation
fluid overload

* Myomectomy \Rightarrow It is enucleation of Myomata from the uterus
leaving behind a potentially functioning organ capable
of Future Reproduction

INFECTIONS

Bacterial vaginosis

- all causing vaginal discharge/vaginitis
- M/c - Bacterial vaginosis > Candida > Trichomonas

- Giardia
vaginalis
(Mycoplasma
Ureaplasma
Mobiluncus)

Doderline replaced
by Giardia.

Candida albicans

T. vaginalis

After passing urine ; Pain left
blc of excitation of skin

- Foul Smelling discharge
itching (X)
dyspareunia (X)
vaginal discharge

- Pruritis
- Discharge may present
- Urinary Symptom (SPLASH DISORDERS)

- Discharge
± dyspareunia
± urinary symptoms
± asthma

Discharge \Rightarrow Foul Smelling

Off white / Greyish

Curdy

Green

White

(Yellow)

pH > 4.5

< 4.5

> 4.5

- IOC - Saline Microscopy

Clue cells \oplus

Pseudo hyphae

organism

see itself

↓

dt + flagella

- Ratio of Poly Morpho Nucleated cells \downarrow
Epithelial cells
 < 1

- Gold Standard - Gram Staining

Nugent Score \Rightarrow 7-10

Culture has No Role in
Bacterial vaginosis

Culture in
SDA Media

Culture in
Stuart's medium

- Amsel's Criteria

If 3 out of 4 - Bacterial
vaginosis

Splash

Dysuria

Strawberry
cervix

i) Foul Smelling (off/white)
discharge

Painful Urination
due to exfoliation of vulva

(Punctate Hemorrhage
grey)

ii) pH > 4.5

Whiff test - \ominus

Whiff test - \oplus

iii) Clue cells (at least 20% of
the epithelial cells)

iv) Whiff test (addition of KOH to D/S \rightarrow Fishy Ammonium)

Not a STD

Usually

Not
STD

STD

Partner Not
treated

Usually Not
(done if partner
is symptomatic)

Yes

**
Doc \Rightarrow Metronidazole

Single dose
fluconazole

Metronidazole

\downarrow
Clindamycin

Can cause Pre-term
Labour in ♀

Recurrent
Vulvo vaginal candidiasis

\Downarrow
≥ 4 episodes / year. **

PID (Pelvic Inflammatory Disease)

Infection of upper genital tract
(uterus / Fallopian tube / ovaries)

M/c organism \Rightarrow Chlamydia
+ Gonorrhoea

age group \Rightarrow 15-25yr

Highest R/F \Rightarrow Multiple sexual partners.

PID in Virgin girl \Rightarrow df. Tuberculosis

PID in GvD users \Rightarrow df. Actinomyces

* Clinical diagnosis : \Rightarrow

\hookrightarrow Pain Lower abdomen & Any of the following

a) Uterine tenderness

b) Cx Motion

c) Adnexal

Ix \Rightarrow * USG \Rightarrow cog wheel sign; Beads on string sign;
waist sign

Endometrial Rx \rightarrow Plasma cell endometritis

\downarrow
Chronic endometritis

\downarrow

Rx of chlamydia

Laparoscopy \rightarrow Best (Gold standard)

\hookrightarrow Fitz-Hugh Curtis Sy.

(191)

FISH - Hugh Carter's Syndrome

↳ Perihepatitis

violin string Adhesions b/w Liver & Anterior
Abdominal wall

Rt. upper quadrant Pain

Liver enzyme (↑)

M/c caused by \Rightarrow Chlamydia > Gonorrhoea

* Long term complication of PID \Rightarrow

- ↳ Infertility;
- ii) Ectopic ♀;
- iii) Chronic Pelvic Pain;
- iv) Recurrent PID;
- v) Hydro salpinges.

CONTRACEPTION

Methods of contraception

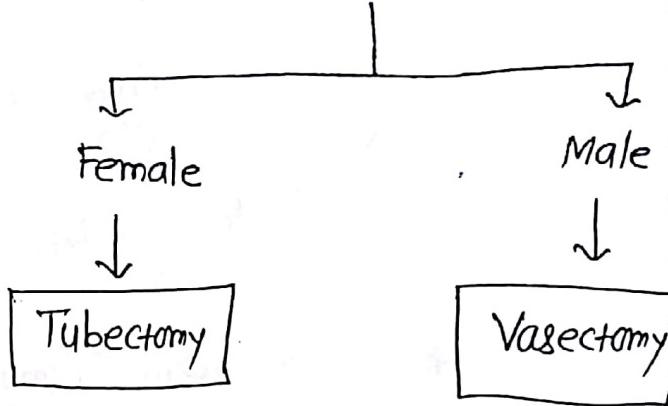
TEMPORARY METHODS

(Used to postpone pregnancy or space birth)

- BARRIER METHOD :
- NATURAL CONTRACEPTION;
- OCPS ;
- Injectables
- Implants
- GUCDs

PERMANENT METHODS

(Aim is to purposefully & permanently destroy the reproductive capacity of an individual).



* OCPs

- * On the basis of Amount of estrogen; they can be classified as →
 - i) Low dose Pills : $< 50 \text{ mcg}$ (Avg: 35 mcg) Ethinyl Estradiol
 - ii) High dose Pills : $\geq 50 \text{ mcg}$ (Ethinyl Estradiol)
 - iii) Very Low dose Pills : $\leq 20 \text{ mcg}$ (Ethinyl Estradiol)
 - iv) Lowest Possible Pills : 10 mcg (Ethinyl Estradiol)
(LoLo Estrogen)

* Mala D & Mala N

- both have 30 mcg ethinyl Estradiol + 0.15 mg Levonorgestrel (LNG)
- Both have 21 hormonal tablets & 7 ferrous fumarate tablets;
- Mala D available @ a cost of 2 Rupees; while Mala N free of cost by Govt. of India.

M.O.A. of OCP \rightarrow Mainly:

Inhibition of ovulation.

* M/c side effect of OCP:

Breakthrough bleeding.

* In Anovulatory SUB:



Estrogen breakthrough bleeding.



Progesterone breakthrough bleeding.

* If a Lady misses 1 pill; take 2 pills + the following doses;
if she misses 2 pills \rightarrow Backup Method of contraception

* In the event of Missing a pill:

Take the Most Recent Missed Pill Immediately; Use condoms for 7 days & continue the packet



Now if

≥ 7 pills are Remaining in packet

Finish the Remaining tablet &
Start the New packet after 7 days gap.

< 7 pills are Remaining in packet

Finish the Remaining pills
& Start the New packet
Next day without a 7 days gap.

Q: A women who is taking Combined OCPs misses 2 consecutive pills.

There are 10 pills Remaining in the packet. Next Pill is

(a) Take both pills immediately continue the packet & use condom for 7 days

(b) Take the Most Recent Missed pill Immediately; continue the packet & use condom for 7 days.

~~(c) Take the Most Recent Missed pill Immediately; continue the packet; use condom for 7 days & commence the next packet after a 7 days gap.~~

* Fertility Return \Rightarrow \exists in 3 Months of withdrawal of the drugs in 90% cases
 \downarrow
(Ovulation Return)

- * Which contraception have least chance of Ectopic $\oplus \rightarrow$ OCP
- * OCP also \downarrow Risk of PID.

But \uparrow Risk of candida & chlamydia are seen by combined oral contraceptives

- * Other M.O.A. of OCPs \Rightarrow Prevention of Fertilization;
Interference \in Implantation;

- * OCP & Cancers \Rightarrow

- OCP & Cervical cancer \rightarrow Yes;

- Ovarian cancer \downarrow by 50%;

- Endometrial cancer \downarrow by 60%;

- Colon cancer \downarrow risk;

- Breast cancer : No Yes Risk

\hookrightarrow OCPs are protective against benign breast disease,
but Role of OCPs are controversial in ca of breast,

- Using HRT (Hormone Replacement Therapy) Yes chance of breast cancer.

- Hepatic Adenoma : Yes Risk;

- HCC : No Yes Risk (Not decrease Risk also);

- Gallbladder cancer ! : No Yes Risk,

* Absolute C/I of OCP (WHO Category 4) \Rightarrow

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(A3)

Mnemonics

- ↳ Banks \rightarrow k/clo Breast Cancer
- Have \rightarrow Severe Hypertriglyceridemia / Hypercholesterolemia
- Various \rightarrow Undiagnosed Vaginal bleeding
- Scheme \rightarrow Stroke; Smoked over age of 35 years.
- To \rightarrow Thrombophlebitis | Thromboembolic disorder
- Provide \rightarrow Pregnancy
- Home \rightarrow Uncontrolled Hypertension ($\geq 160 \text{ mmHg}$)
- Lovins \rightarrow Acute Liver disease (Hepatitis; Cirrhosis)
- During \rightarrow Diabetes & Vasculopathy
- May \rightarrow Migraine & Aura (i.e. Focal Neurological deficit).

also; Coronary Artery Disease is absolute C/I of ocp.

* Newly Married couple : choice of contraception : OCP

Living far apart; Meeting occasionally

↳ Barrier Method

* Safety : Which contraception is safest : "Barrier"

\downarrow
S/E \Rightarrow very high failure rate

\therefore Pregnancy is S/E

PROGESTERIN ONLY PILLS (POP)

aka "Minibills" (75mcg Progesterone)

M.O.A. : Thickening Cervical Mucus

* Should be taken on Same-time Everyday



(Safe Margin < 3hrs)



If delay was for > 3 hrs - Back Method Should be used

• POP's all contraception of choice in Lactating Female



↳ can be started immediately after delivery, POP > gUD

* Contraception of choice in Lactating's Female : POP > gUCD

↳ but Not Lactating Amenorrhea

↳ b/c high failure Rate

* Minifill available in India → "CERAZETTE"



M.O.A. → Mainly Again



Inhibition of ovulation

Safe Margin : 12 hrs

(A delay of upto 12 hrs can be accepted)

* M/c Side effect of POP : Irregular vaginal bleeding.

* Absolute CI : i) Pregnancy;

ii) Undiagnosed vaginal bleeding; } Same for all
iii) K/c/o Breast Cancer }

PROGESTERONE INJECTIONDMPA (Depot Medroxyprogesterone Acetate)Net En (Norethisterone enanthate)Dose \Rightarrow 150 mg ilm

200 mg ilm

To be Repeated every 3 Months

Repeated every 2 Months

Pt. can wait up to 4 weeks late
for Next injectionPt. can wait up to 2 weeks
Late

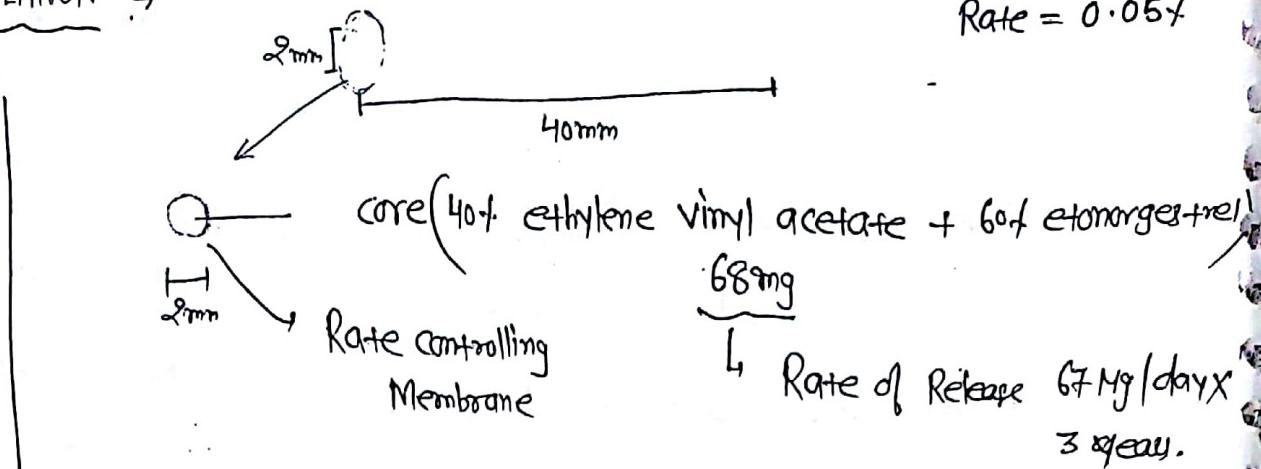
- * DMPA \downarrow 2 Good things \Rightarrow
 - i) Useful in Female \in Epilepsy
 \uparrow Seizure threshold;
 - ii) Reducing Sickling crisis: Useful in Sickle cell Anemia;

2 bad things \Rightarrow i) Significant Bone loss;ii) Delay in Return of Fertility;
Avg. delay : 12 MonthsMax^m. delay : 18 Months.* M/c side effect of DMPA : Irregular Vaginal Bleeding.* Absolute C/I : Same three SIE

- L Undiagnosed vaginal bleeding
- Pregnancy
- K/c/o Breast cancer

PROGESTIN ONLY IMPLANTS \Rightarrow Among all contraceptives; Least failure Rate = 0.05%

IMPLANON \rightarrow



Single Rod \equiv keto-desogestrel

OPD Procedures (Most Popular Implant Now-a-days):

M.O.A. \Rightarrow Inhibition of ovulation.

Site of Implantation \Rightarrow Non-dominant Arm (Medial aspect of Upper Arm).

• gt is Not Radio opaque.

∴ Next Generation of IMPLANON: EDEXPLANON

↳ Radio opaque

M/c side effect: Irregular vaginal bleeding.

Absolute C/I: Same 3

i) Pregnancy

ii) Undiagnosed vaginal bleeding

iii) K/c/o Breast cancer

NUVA

• gt is Vaginal Ring (Blue or white);

• It is E+P compound ($E = \text{Ethynodiol Diacetate} : 15 \text{ mg/day}$)
 $P = \text{Etonogestrel} : 120 \text{ mg/day}$)

M.O.A.: Inhibition of ovulation.

How to Use : Insert itself in vagina on 1st day of her Menstrual cycle & keep it for 3 weeks. After 3 weeks ; Last week is "Ring free" (195)



then : Insert New Ring.

Safe period of Ring : 3 hours before sex.

It means if the expelled NuvaRing has been out of vagina for more than 3 continuous hours during weeks 1 & 2; you may not be protected from ♀.

INTRAUTERINE DEVICES

1st Generation IUD's

Insert or Non-Medicated Devices

eg ⇒ Lippes Loop

↳ Inserted by

"Push out technique".

Copper wire

• CUT 380 A

• Release Rate : 50 Mg/day

• Radio opaque : Coated w/ Barium

• Free of cost by govt. of India

2nd Generation IUD's

It consists of copper or silver containing IUD's

eg ⇒ CUT-220C; CUT-380A

NovaT; Multiload Cu 250/375

→ Klas "PARAGUARD". ***

CUT 380 A

Shate

Arms (i.e. ***

Copper wire is also found on Arms

Surface Area

• MIRENA (aka LNG 20

LNG-52mg

20 Mg/day

Radio opaque

• Not free (700/-)

CUT 380A

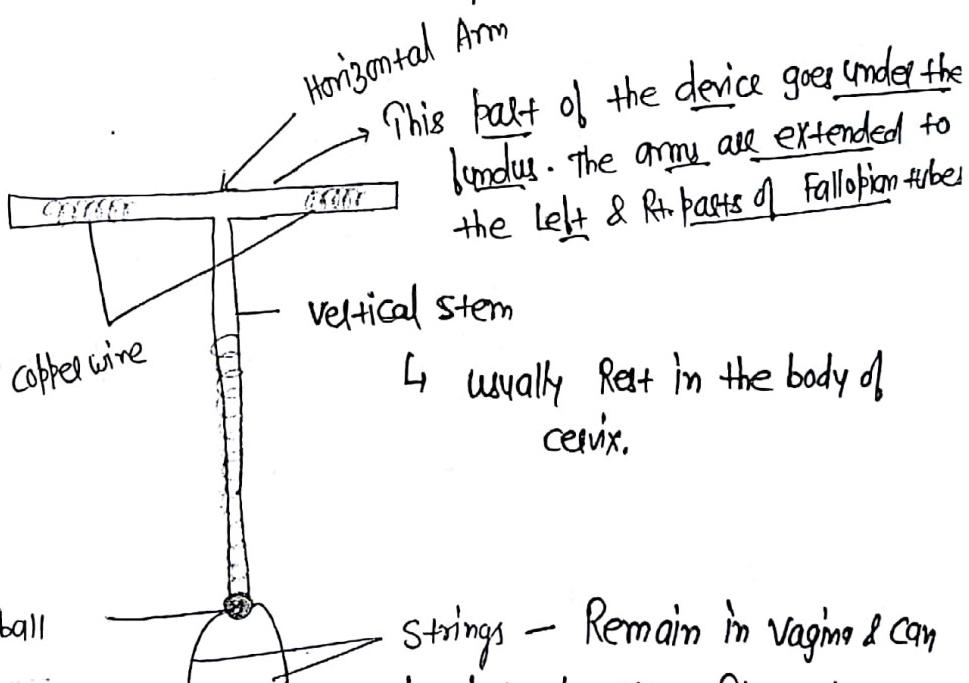
Mlrenq

- Effective for span of 10 years;
- presence of break : to Ver Risk of Perforation & for Identification and Removal.
- Can be used as Emergency contraceptives
- For Menstrual blood Loss
- * M/c Side effect of GUD \Rightarrow ↑ Bleeding;
M/c cause for Removal \Rightarrow Pain
of GUD
- * G.U.D. : Mechanism of Action \Rightarrow Mainly "Spermicidal"
Ans \Rightarrow Inhibition of Fertilization
Inhibition of Implantation
- M/c Infection of GUD : Actinomycetes;
- * GUD In situ \bar{c} ♀ : Remove GUD
Why
b/c Risk of Abortion
- * Do USG b/c failure of GUD or ♀ \bar{c} G.U.D. Mostly ; Ectopic*
- * Mark Infection \bar{c} GUD : Within 20 days of insertion*
- * Multifilamentous : Olden days \rightarrow ↑ Risk of Infection;
- * Monofilamentous : Now a days \rightarrow ↓ Risk of Infection \rightarrow that too by dogon

Misplaced GUD (Missing thread) \rightarrow G.O.C \Rightarrow USG
 \downarrow If Not found
X-Ray (196)

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- * GUD Missing \in Perforation \oplus in Abdomen: Mx: Laparoscopy**
- * If GUD Embedded in Myometrium: Mx: Hysteroscopy & Remove it.
- * Contraception in HIV+ Patient \Rightarrow GUD + Barrier
- * D.M. \Rightarrow GUD
- * Heart disease \Rightarrow GUD
- * M/c Mode of contraception used in India: Barrier Method*
- * Best contraception; if Family is complete: Vasectomy $>$ Sterilization $>$ GUD $>$ OCP
- * Best Non-permanent contraception; if Family is complete: GUD $>$ OCP.*
- * No Risk of Teratogenicity; if a ♀ Female continue ♀ \in GUD



Strings - Remain in Vagina & can be felt by User. These days Monofilament goco are available

CU-T 380A

Silver Line CU 380 Ag \Rightarrow M/c CU T used worldwide

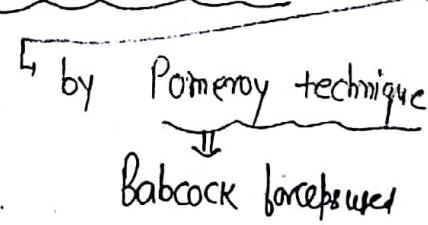
↳ have flexible Arms: Silver core (T) & Rest same as CUT 380A

* Absolute C/I of GUCD - category 4 of WHO ↗

- Mnemonic → Please → Puerperal Sepsis or Postabortal sepsis
Don't → DUB / Unexplained vaginal bleeding
Try to → Gestational Trophoblastic Disease
Put → Current PID / STD or C/ in Past 3 Months
condom → Puerperal Sepsis, know Pelvic TB
→ Ca Cervix
→ Ca endometrium

STERILIZATION (Permanent Method)

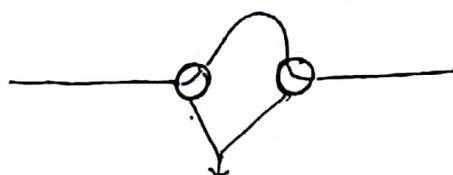
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- * No. of children Required for sterilization ?? 197
Ans ⇒ At least 1 child of 1 yr old.
- * If it is a legal procedure → Consent is Mandatory; but only of Client.
* Consent of spouse is Not Mandatory.
- 22 yr - 45 yr can undergo sterilization;
- Q. UnMarried women can undergo sterilization ?? ⇒ NO
Married or even married ⇒ Yes
- Most cost effective Mode of contraception : Vasectomy
- Most effective contraception : Implant
- How Many days after vasectomy should avoid : > 3 Months till coitus or use another contraception Azospermia
- Postpartum Sterilization ⇒ Within 7 days of delivery ①
after 42 days of postpartum (Can't do ② 8th month — days;
• This is known " Interval sterilization."
- M/C Method used for Postpartum sterilization : Minilabotomy (3 cm Long)


by Pomeroy technique
↓
Babcock forceps
- We don't use Laparotomy / Laparoscopic sterilization in Post-partum sterilization.
- * Post-placental gud ⇒ if we put gud \leq 10 min of delivery;
- * Post-partum gud ⇒ if we put gud \leq 1 hr of delivery,

- * M/c part of Fallopian tube that we ligate in sterilization
↳ Gsthamus.

Modified Pomeroy
aka "Parkland Method"



double ligation of tube is done

Failure Rate = 0.2

Pomeroy



Middle part of tube (3-4 cm from fundus) is formed into loop using Babcock forceps; which is tied & excised 0.4

- * Interval sterilization → Non-pregnant State

Whenever we do sterilization in Non-♀ Female is k/a as "interval"

M/c Method used : Laparoscopy

- * M/c Method of Female Sterilisation ⇒ Laparoscopic sterilisation,

CO₂ gas used

↓
Intraabdominal Pressure b/w 8-12 mm ; Maxmⁿ = 15 mm of Hg
Not More than 15 mm of Hg b/w it else

- * M/c Method for Laparoscopic Ligation ⇒ Yoon balloon Ring / ^{Venous Return} Sigmoidic band
- * Site of Ligation ⇒ Gsthamus

- * Among sterilization technique ⇒

Least failure Rate : Unipolar cautery > Modified Pomeroy
(Never Used)

↳ b/w of Inter-tissue b/w

Highest failure Rate : Clip > Bipolar cautery

↓
HULK CLIPS

Reversal Methods : Clips > Falope Ring > Modified Pomeroy ³⁹³ ~~Cautery~~

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Laparoscopic Ligation → Pt. In Lithotomy position

↓
With help of Veres Needle (Introduced at
an Angle of 45°) Pneumoperitoneum is created
↓ \hookrightarrow CO_2 gas used

Procedure is done under Sedation & Local
Anesthesia

- * M/c wed Method in Minilaparotomy : Modified Pomeroy.
- * Highest Risk of ectopic ♀ : Cautery > Madlenai > Modified Pomeroy.

